JOURNAL

OF THE

American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.

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Vol. LXXV, N. S. Vol. 28 August, 1929 No. 2

ON TO DETROIT

Plans for the Detroit meeting are practically complete. Only a few of the details remain to be worked out by the Local Committee on Arrangements. Only those who have been behind the scenes and participated in planning for an A. V. M. A. convention can have the proper conception of the responsibilities which are placed on the shoulders of the veterinarians located in the convention city. Our annual gatherings have been growing in size, all attendance records having been broken at Minneapolis last year, with a registration in excess of 1500.

Being international in scope, the A. V. M. A. conventions move around the United States and Canada, with the result that one city does not have an opportunity to entertain the A. V. M. A. more often than once in a decade or longer. This year will be the third time that an A. V. M. A. convention has been held in the Motor City, the previous meetings having been held in 1900 and 1916. One result of this interval between meetings in the same city is the change in the personnel of the profession locally. Only on rare occasions is there anybody who has had the benefit of previous esperience in entertaining the A. V. M. A. Of the nine men who constituted the Local Committee on Arrangements for the Detroit meeting in 1916, four have died and three have removed from the City.

Philadelphia has had three meetings during the past quarter-century—1908, 1918 and 1927. Kansas City has had two—1907

and 1917. Minneapolis was a repeater, but at a twenty-six year interval, 1902 and 1928. Canada has had three meetings: Ottawa, in 1903; Toronto, in 1912, and Montreal, in 1923. St. Louis has had two, in 1904 and in 1922. New York has not had a meeting since 1913 and Chicago none since 1909. California has had two conventions—at San Francisco, in 1910, and at Oakland, in 1915. The meeting at New Orleans, in 1919, was the second to be held in the South, the first having been in Nashville, in 1897. Lexington had the 1926 convention. The Pacific Northwest was visited the first time in 1925, at Portland. The meeting at Des Moines, in 1924, was the second in the Iowa capital, the first having been in 1895. Cities that have had one meeting are: Buffalo (1896), Omaha (1898), Atlantic City (1901), Cleveland (1905), New Haven (1906), Indianapolis (1912), Columbus (1920), and Denver (1921).

On to Detroit!

Donation

Dr. L. E. Willyoung, of Princeton, W. Va., has donated to the A. V. M. A. library forty-four bound volumes of several veterinary periodicals, including the *American Veterinary Review*, *Journal of Comparative Medicine and Veterinary Archives* and Proceedings of the United States Veterinary Medical Association and American Veterinary Medical Association. In addition to the bound volumes, there were about fifty-odd numbers of different veterinary journals. On behalf of the Association, we make this acknowledgement and thank Dr. Willyoung for his kindness.

A section of Detroit's sky-line

JUST ABOUT RABIES

Erroneous statements concerning rabies are so frequently seen in the newspapers nowadays that they have become rather commonplace. However, when we see one of these misstatements in a highly reputable medical journal, it calls for some comment. Several months ago, an editorial in one of our medical journals contained the following statement:

> It occurs to us that there are many stray dogs in the country, and they are always a menace. They may be afflicted with skin diseases, infested with vermin, or in other ways unclean or diseased. And these dogs may under certain conditions develop rabies at any time.

There is a strong suggestion here that the writer of this editorial holds the belief that rabies is a non-infectious disease, one that can develop spontaneously, *under certain conditions*, whatever these conditions may be. There is a strong inference in another paragraph of the editorial, that rabies is a "hot weather" disease.

Even Arthur Brisbane, who writes on about everything under the sun, and who, it might be mentioned in passing, has no particular love for dogs, displays his lack of knowledge of scientific progress when he laments:

> A new vaccine prevents distemper in dogs absolutely, "all cases, all breeds."
>
> That's not important news, except to dogs. A vaccine that would keep dogs from going mad and biting children would be more valuable.

Evidently Mr. Brisbane does not know that for five years or more veterinarians have been doing the very thing that he so much desires—protecting children (and grown-ups, too, as well as animals) against rabies by vaccinating dogs against the disease.

As an illustration of how newspapers some times mix truth with fiction, in discussing rabies, we quote an editorial from the *Detroit Free Press*

> The increase of rabies in Detroit which the department of health reports is inexcusable. It is a direct result of neglect. For rabies can be eliminated in a city, or at the worst it can be confined to occasional, sporadic cases.
>
> The measures to be taken are simple. They consist of the rigid enforcement of an ordinance providing that no unmuzzled dogs are to run at large. Possibly it is reasonably safe to permit an exception in favor of vaccinated animals, but there are increasing indications that vaccination is not a perfect protection against disease, although it appears that a bite from a mad animal that has been vaccinated is less virulent than a bite from a mad animal that is unvaccinated and that the efficacy of the Pasteur treatment is more certain in case infection comes from an animal in the first classification.
>
> For a time an ordinance providing that all dogs must be kept confined or in leash was in effect in Detroit and was enforced with some energy. Rabies disappeared. The ordinance went into the discard. Rabies began to spread. It is still spreading.

At the regular monthly meeting of the Wayne County (Mich.) Medical Society, December 4, 1928, the members participated in a symposium on rabies. In a paper presented by Dr. W. M. Donald, one of Detroit's foremost clinicians, the following statement was made:

> . . . my attention has been directed to this subject (rabies) at varying intervals through my association with laboratory men, veterinarians, and public health workers, to all of whom I owe much. From them, I have learned practically all that I know of this disease, the ordinary physician being entirely ignorant of the whole matter.

Later in his address, Dr. Donald paid a nice tribute to the veterinary profession, as follows:

> The experience of Great Britain and Prussia, which I have mentioned, and which has been duplicated by other countries since, would lead us to believe that the forced muzzling of dogs and the destruction of all street curs unattached and unclaimed would be of sweeping value. Public health certainly is in the saddle in this year of our Lord, and it behooves us of the medical profession generally to stand solidly back of public health control in this, as in other communicable diseases of this type. Our friends, the veterinarians, are entitled again to our admiration for their constant efforts at the control of this and similar diseases in the animal kingdom.
>
> We physicians owe both these groups our aid, and it is not inconceivable to my mind that a great society like this could do much towards showing the world, by its constant support of public health and veterinary measures, that a practical elimination of this disease is feasible.

Among those who participated in the symposium was Dr. Herbert W. Emerson, Director of the Pasteur Institute, University of Michigan, Ann Arbor. In his remarks on rabies Dr. Emerson made a point that many veterinarians can well appreciate. He said:

> It is difficult and sometimes impossible to enforce proper restrictions on dogs because of the ignorance and carelessness of the public, and because of the shortsightedness and selfishness of certain organizations, chief among which are the "kennel clubs." These organizations claim to be interested in dogs, but are usually dominated by those members making money out of raising and selling dogs, or journals about dogs. These organizations have been the greatest obstacles to the enforcement of proper regulations.

The symposium was rounded out by contributions by two veterinarians, Dr. B. J. Killham, State Veterinarian, and Dr. A. S. Schlingman, of Detroit. The former spoke on rabies from the standpoint of a sanitary control official, and the latter on the various types of antirabic vaccines. Attention is being directed to the value of meetings of this kind, where veterinarians may meet physicians and discuss problems of mutual interest. Such contacts can not be anything but helpful to both professions.

Detroit has been under three flags—American, French and British—two of them twice, the French and the British.

ADDITIONAL PROGRAM MATERIAL

The following papers have been secured for the Detroit meeting, in addition to those listed in the July issue of the JOURNAL:

GENERAL SESSION

The State's Obligation to the Poultry Industry—James E. Rice, Professor of Poultry Farm Management, Cornell University, Ithaca, N. Y.

SECTION ON GENERAL PRACTICE

Feeds and Feeding—Dr. Carl W. Gay, Ohio State University, Columbus, Ohio.

SECTION ON SANITARY SCIENCE AND FOOD HYGIENE

Avian Tuberculosis in Nebraska—Dr. G. H. Hays, State Veterinarian, Lincoln, Nebr.

SECTION ON SMALL ANIMALS

Immunization of Dogs Against Distemper by the Laidlaw-Dunkin Method—Dr. A. Eichhorn, Pearl River, N. Y.

The Michigan State Department of Health protects visitors to Michigan by very careful sanitary inspection of resorts.

Book-Cadillac Hotel

A. V. M. A. SECTION CHAIRMEN 1928-29

DR. WM. A. BELL
Nashville, Tenn.
General Practice

(above)

(below)

DR. CHAS. W. BOWER
Topeka, Kan.
Small Animals

DR. H. E. BIESTER
Ames, Iowa
Education and Research

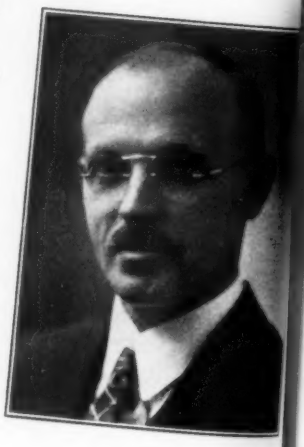
(above)

MAJOR R. A. KELS
Washington, D. C.
Sanitary Science and H
Hygiene

(above)

(below)

DR. H. J. STAFSETH
East Lansing, Mich.
Poultry



A. V. M. A.
SECTION
SECRETARIES
1928-29

DR. E. P. ALTHOUSE
Sunbury, Pa.
General Practice

(above)

(below)

DR. H. J. MILKS
Ithaca, N. Y.
Small Animals

DR. F. H. BROWN
Indianapolis, Ind.
Sanitary Science and Food Hygiene

(above)

(below)

DR. R. O. BILTZ
Harrisburg, Pa.
Poultry

DR. W. A. HAGAN
Ithaca, N. Y.
Education and Research

(above)

AT DETROIT IN 1900

The official records of the American Veterinary Medical Association show that fifty-six members and thirty-three members-elect attended the meeting held in Detroit in 1900. Of the fifty-six members the following are still on the roll:

Dr. E. B. Ackerman
Dr. A. H. Baker
Dr. Tait Butler
Dr. William H. Dodge
Dr. Joseph Hawkins
Dr. G. A. Johnson
Dr. Wm. Henry Kelly
Dr. Wm. Herbert Lowe
Dr. W. J. Martin
Dr. Robert C. Moore
Dr. E. M. Nighbert
Dr. E. C. Porter
Dr. Leon N. Reefer
Dr. James Robertson
Dr. Walter Shaw
Dr. T. E. Smith
Dr. W. L. Williams

The following members-elect, still on the roll, were at the Detroit meeting:

Dr. F. E. Anderson
Dr. R. A. Craig
Dr. Langdon Frothingham
Dr. H. Fulstow
Dr. H. M. Gohn
Dr. William S. Hiday
Dr. C. A. Miller
Dr. F. P. Ruhl

The total attendance was 283, including ladies and visitors.

Dr. Theobald Smith, who has been director of the Department of Animal Pathology, Rockefeller Institute for Medical Research, since July 1, 1914, retired June 30, 1929. Dr. Carl Ten Broeck is acting director.

Dr. William H. Welch, of Johns Hopkins University, Baltimore, retired as president of the Maryland State Board of Health, after having been connected with the Board for 31 years.

The Coliseum, State Fair Grounds, Detroit, where the clinic will be held, August 16.

APPLICATIONS FOR MEMBERSHIP

(See July, 1929, JOURNAL)

FIRST LISTING

ACKERMAN, NORMAN W. Van Horne, Iowa
D. V. M., Iowa State College, 1915
Vouchers: J. C. Glenn and Grant B. Munger.

ALLEN, GEORGE HENRY 1125 N. Madison St., Dallas, Texas
D. V. S., Kansas City Veterinary College, 1911
Vouchers: N. F. Williams and W. R. McCuistion.

ANDRIES, ALBERT JOSEPH Box 145, Deloraine, Man.
V. S., Ontario Veterinary College, 1919
Vouchers: J. B. Still and M. J. Kellam.

APPLEWHITE, THOMAS HENRY 339 W. 26th St., Jacksonville, Fla.
D. V. M., Indiana Veterinary College, 1914
Vouchers: R. L. Brinkman and T. W. Cole.

BARNES, ALLEN S. 513 Logan St., Frankfort, Ky.
D. V. M., Cincinnati Veterinary College, 1911
Vouchers: W. F. Biles and D. E. Westmoreland.

BECKER, L. H. Bellevue, Iowa
D. V. M., Chicago Veterinary College, 1912
Vouchers: Grant B. Munger and John B. Bryant.

BENSON, DAVID ALBIN 5924 S. Richmond St., Chicago, Ill.
D. V. M., Grand Rapids Veterinary College, 1918
Vouchers: A. J. Noonan and H. Busman.

BRIGGS, LYLE HOWARD 459 Prentis St., Detroit, Mich.
D. V. M., Michigan State College, 1926
Vouchers: Hugo Cornehl and H. Preston Hoskins.

BROWN, LAURISTER NASH 441 Turner St., Auburn, Maine
B. V. Sc., Ontario Veterinary College, 1929
Vouchers: L. K. Green and G. R. Caldwell.

CARVER, H. C. Higginsville, Mo.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: J. D. Ray and A. T. Kinsley.

CASEY, JOHN WILLIAM 3690 Oak St., Jacksonville, Fla.
D. V. M., Cornell University, 1913
Vouchers: T. W. Cole and B. N. Lauderdale.

CHASE, CARL EDDRIE 286 Pleasant St., Concord, N. H.
B. Sc., University of New Hampshire, 1925
D. V. M. Ohio State University, 1928
Vouchers: F. F. Russell and R. W. Smith.

CHERRY, JOHN L. Tarkio, Mo.
D. V. S., U. S. College of Veterinary Surgeons, 1913
Vouchers: J. D. Ray and A. T. Kinsley.

CLARKE, ROY E. R. R. No. 1, Box 52 D, Montesano, Wash.
D. V. M., State College of Washington, 1929
Vouchers: James H. Woodside and E. E. Wegner.

COOKE, HERBERT T. B. 1304 N. 28th St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1911
Vouchers: G. A. Dick and H. E. Bemis.

COTTON, DANIEL LUZERN Beresford, S. Dak.
D. V. M., Chicago Veterinary College, 1918
Vouchers: H. J. McCauley and C. C. Lipp.

CRIDER, R. A. Elgin, Iowa
D. V. M., Iowa State College, 1920
Vouchers: Grant B. Munger and John B. Bryant.

New Hill Memorial Library
Louisiana State University

CROOKS, HARRY F. Chilhowee, Mo.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: J. D. Ray and A. T. Kinsley.

CROUCH, SAM 816 So. San Pedro St., Los Angeles, Calif.
D. V. M., McKillip Veterinary College, 1920
Vouchers: W. L. Curtis and L. J. Cook.

DAVIS, ART MARSHALL Akron, Iowa
D. V. M., Iowa State College, 1917
Vouchers: C. H. Stange and C. H. Covault.

DROMGOOLE, E. B. 1314 Texas Ave., Mart, Texas
D. V. M., Southwestern Veterinary College, 1912
Vouchers: N. F. Williams and H. L. Darby.

EDELIN, ALLEN A. Laurel, Md.
D. V. M., United States College of Veterinary Surgeons, 1914
Vouchers: E. M. Pickens and E. B. Simonds.

EDMISTON, GEO. A. H. Easton, Md.
D. V. M., McKillip Veterinary College, 1914
Vouchers: E. M. Pickens and J. P. Turner.

FARNSWORTH, GEORGE K. 2031 Cambridge St., Los Angeles, Calif.
V. S., Ontario Veterinary College, 1885
Vouchers: W. L. Curtis and L. E. Pike.

FAUKS, CARL H. 1919 W. Ash St., Oklahoma City, Okla.
M. D. V., McKillip Veterinary College, 1907
Vouchers: L. J. Allen and A. H. Denham.

FLAXMAN, CHARLES E. Darien, Conn.
V. M. D., University of Pennsylvania, 1925
Vouchers: M. R. Powers and C. O. Neuhaus.

GIBBS, CLARENCE J. 326 E. St. N. E., Washington, D. C.
D. V. M., United States College of Veterinary Surgeons, 1925
Vouchers: E. M. Pickens and E. B. Simonds.

GILLOON, T. J. Dyersville, Iowa
D. V. M., McKillip Veterinary College, 1908
Vouchers: Grant B. Munger and John B. Bryant.

GOW, ALEXANDER, JR. New York State Veterinary College, Ithaca, N. Y.
D. V. M., Cornell University, 1929
Vouchers: Herbert L. Gilman and E. Sunderville.

HATCHER, NELSON N. Clinton, La.
D. V. M., St. Joseph Veterinary College, 1920
Vouchers: Hartwell Robbins and E. Heiny.

HENDERSON, FINIS E. 643 Court St., Elko, Nev.
D. V. M., Kansas State Agricultural College, 1929
Vouchers: Edward Records and W. H. Hilts.

HOLLISTER, A. V. Clinton, Wis.
M. D. V., McKillip Veterinary College, 1907
Vouchers: James S. Healy and H. D. Larzelere.

HORN, HOMER H. 2309 S. 50th Ave., Cicero, Ill.
D. V. M., McKillip Veterinary College, 1908
Vouchers: L. Enos Day and H. Busman.

HOULDSWORTH, JOSEPH DEAN 180 Green Lane, Manayunk, Pa.
V. M. D., University of Pennsylvania, 1892
Vouchers: C. O. Neuhaus and Louis A. Klein.

INGRAM, WILLIAM L. Opelika, Ala.
D. V. M., Alabama Polytechic Institute, 1910
Vouchers: W. B. Fleming and D. J. Meador.

JONES, EVERETT B. Box 702, Vernon, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: Frank D. Porter and N. F. Williams.

KELLOGG, LOUIS WARREN — Anamosa, Iowa
D. V. M., Chicago Veterinary College, 1917
Vouchers: J. C. Glenn and John B. Bryant.

KNOTTS, FRANK RANKINS — 1101 Duncan St., Stillwater, Okla.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: C. R. Walter and C. H. McElroy.

LADSON, HERMAN H. — R. F. D. 5, Rockville, Md.
D. V. S., United States College of Veterinary Surgeons, 1911
Vouchers: E. M. Pickens and L. J. Poelma.

LAWHON, GLEN J. — Hartsville, S. C.
D. V. M., McKillip Veterinary College, 1917
Vouchers: M. R. Blackstock and J. P. Caughman.

LEE, WILLIAM J. — 39th St. and Woodland Ave., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1909
Vouchers: G. A. Dick and Louis A. Klein.

LENTZ, FRANK E. — 39th St. and Woodland Ave., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1907
Vouchers: G. A. Dick and C. O. Neuhaus.

LEWIS, HENRY V. — State College Station, Brookings, S. Dak.
D. V. M., Iowa State College, 1929
Vouchers: W. F. Guard and H. D. Bergman.

LEWIS, LAWRENCE — Clarksville, Texas
M. D. C., Chicago Veterinary College, 1909
Vouchers: Jesse L. Shabram and N. F. Williams.

LEWIS, LAWRENCE JOHN — 330 Federal Bldg., Madison, Wis.
D. V. M., Ohio State University, 1916
Vouchers: James S. Healy and H. D. Larzelere.

LOVE, JAMES ROBERT — Elliott City, Md.
D. V. M., Ohio State University, 1907
Vouchers: E. M. Pickens and E. B. Simonds.

LOWELL, J. A. — 117 S. Bell, Shawnee, Okla.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: C. R. Walter and C. Pedrick.

MCCLUNG, HUGH EDWARD — 831 Milford, Glendale, Calif.
D. V. M., Kansas State Agricultural College, 1929
Vouchers: W. L. Curtis and G. W. Blanche.

MCDERMONT, G. F. — 1932 So. Hobart Blvd., Los Angeles, Calif.
V. S., Ontario Veterinary College, 1910
Vouchers: W. L. Curtis and L. M. Hurt.

MILLER, CHAUNCEY BRADY — 1001 Ocean Ave., San Francisco, Calif.
M. D. C., Chicago Veterinary College, 1910
Vouchers: Joseph M. Arburua and Oscar J. Kron.

MOE, PAUL S. — 2329 Grant Ave., Fresno, Calif.
D. V. M., Colorado Agricultural College, 1926
Vouchers: A. C. Rosenberger and D. R. Cook.

NIEMANN, KARL WILLIM — 31 W. 9th St., Reno, Nev.
D. V. M., Kansas State Agricultural College, 1929
Vouchers: Edward Records and Lyman R. Vawter.

ORMISTON, ADAM W. — 6353 McCallum St., Germantown, Philadelphia, Pa.
D. V. S., American Veterinary College, 1892
Vouchers: G. A. Dick and C. O. Neuhaus.

PARROTT, WALTER L. — U. S. Bureau of Agricultural Economics, Dover, Del.
D. V. M., Kansas State Agricultural College, 1926
Vouchers: Frank Hare and C. C. Palmer.

PECK, EDWIN L. — New Augusta, Miss.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: S. J. Horne and Hartwell Robbins.

PHILLIPS, FRANCIS M. 506 5th St., Dell Rapids, S. Dak.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: M. W. Ray and C. C. Lipp.

PILGRIM, ERNEST C. 214 S. Porter, Okmulgee, Okla.
D. V. M., Kansas City Veterinary College, 1914
Vouchers: C. R. Walter and C. Pedrick.

ROGERS, ROBERT LEE, JR. Health Department, City Hall, San Angelo, Texas
D. V. M., A. & M. College of Texas, 1925
Vouchers: N. F. Williams and H. L. Darby.

RUSH, WILLIAM MARVIN Box 202, Wink, Texas
D. V. M., Southwestern Veterinary College, 1913
Vouchers: H. L. Darby and N. F. Williams.

SCHEIDY, SAMUEL FRANKLIN 39th St. & Woodland Ave., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1929
Vouchers: G. A. Dick and E. L. Stubbs.

SCHONDAU, THEO. Claremont Hotel, Baltimore, Md.
D. V. S., United States College of Veterinary Surgeons, 1910
Vouchers: E. M. Pickens and E. B. Simonds.

SHAW, RALPH ROBERT St. Paris, Ohio
D. V. M., McKillip Veterinary College, 1916
Vouchers: L. A. Mosher and C. E. Inskeep.

STEPHENSON, OSCAR 607 E. Broadway, Sweetwater, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: H. L. Darby and N. F. Williams.

STEWART, RAY ELLIOTT Riceville, Iowa
D. V. M., Colorado Agricultural College, 1921
Vouchers: W. L. Dockstader and Chris. E. Juhl.

STRAIN, C. B. Dunkerton, Iowa
D. V. M., McKillip Veterinary College, 1913
Vouchers: Grant B. Munger and John B. Bryant.

STRODE, CLIFTON W. Ash Grove, Mo.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: A. T. Kinsley and A. Trickett.

TREMAN, PERRY E. Odebolt, Iowa.
D. V. M., Iowa State College, 1919
Vouchers: Guy S. Jones and John B. Bryant.

VOLLSTEDT, WILLIAM C. Dixon, Iowa
M. D. C., Chicago Veterinary College, 1911
Vouchers: Grant B. Munger and John B. Bryant.

WARD, JOHN W. 1638 Westheumer St., Houston, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: N. F. Williams and H. L. Darby.

WATSON, CHARLES EDWIN Lafayette Hill P. O., Pa.
V. M. D., University of Pennsylvania, 1929
Vouchers: E. L. Stubbs and G. A. Dick.

WEBSTER, CHARLES Great Bend, Kansas
D. V. M., Colorado Agricultural College, 1914
Vouchers: J. H. Cheney and N. L. Townsend.

WELSH, MARK FREDERICK College Park, Md.
D. V. M., Michigan State College, 1919
Vouchers: E. M. Pickens and L. J. Poelma.

WESTERBERG, HERMAN P. Simsbury, Conn.
B. V. Sc., Ontario Veterinary College, 1924
Vouchers: George E. Corwin and Fred J. Brockett.

WHEELER, N. M. Winnsboro, Texas
D. V. M., Southwestern Veterinary College, 1912
Vouchers: N. F. Williams and H. L. Darby.

WILSON, JOHN T. Pawnee, Okla.
D. V. M., Kansas State Agricultural College, 1910
Vouchers: C. R. Walter and C. Pedrick.

YOUNG, HARVEY WILSON 3634 Main St., Kansas City, Mo.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: J. D. Ray and A. T. Kinsley.

Aplications Pending

SECOND LISTING

Bailey, William Westley, Box 1092, Red Lodge, Mont.
Barney, Joseph Clement, Magnolia, Miss.
Barry, James R., Wonewoc, Wis.
Beretta, Edward H., Cresco, Iowa
Blackburn, Herbert Lawrence, 713 N. Main, Fort Worth, Texas.
Bolin, Fonsoe M., Turtle Lake, Wis.
Bott, Thomas L., Coldwater, Mich.
Boyce, Harold J., 610 Sandusky St., Kansas City, Kans.
Boyd, Benj. W., Box 462, San Antonio, Texas.
Boyer, Theodore Ross, 1663 21st Ave., San Francisco, Calif.
Brooks, Clarence Shepard, 304 Mapleton Ave., Hollister, Calif.
Brown, Wilbur L., 531 5th St., Fowler, Calif.
Chandler, Guy D., 4160 Ellis Ave., Chicago, Ill.
Chappell, Robert Forrest, Navasota, Calif.
Coffland, W. T., 895 Mar Vista Ave., Pasadena, Calif.
Cole, Alonzo B., New Mulford, Pa.
Cooke, Wm. A. Jr., 2057 Parrish St., Philadelphia, Pa.
Cote, Frank J., 300 Eramosa Road, Guelph, Ont.
Covington, C. R., 701 Wheat Bldg., Fort Worth, Texas.
Eastman, Donald A., 864 A Ave., Cedar Rapids, Iowa.
Edelman, Frank J., 151 Main St., Bath, Pa.
Elliott, John Wilfred, 310 S. 2nd St., Aberdeen, S. Dak.
Ervin, William J., 117 Beechwood Ave., Pawtucket, R. I.
Ewers, James E., 300 State House, Phoenix, Ariz.
Ewers, Smith V., c/o Arizona Packing Co., Phoenix, Ariz.
Farrell, Joseph Milton, 1509 Speedway, Wichita Falls, Texas.
Fickensher, Harry J., 1534 E. 67th Place, Chicago, Ill.
Fitzpatrick, Dennis B., Falfurrias, Texas.
Gaw, Hugh, 46 Barrows St., N. Attleboro, Mass.
Geick, Walter L. G., 709 Fowler St., Waterloo, Iowa.
Green, Fred B., Lufkin, Texas.
Gregory, Rece Carnell, Georgetown, Texas.
Griffey, James Homer, 16 Highland Ave., National City, Calif.
Grover, Capt. Sawyer Adelbert, Fort Howard, Md.
Harrington, Charles F., 933 E. Fifth Ave., Mitchell, S. Dak.
Hartnell, Berton Thomas, Stacyville, Iowa.
Hawkins, J. T., Tyler, Texas.
Hodges, W. Ross, Ranger, Texas.
Hover, Eugene, Box 837, Reno, Nev.
Hylton, Floyd D., Box 90, Capitol Station, Helena, Mont.
Kintner, Capt. John H., Fort Sill, Okla.
Kitchen, Frank Elliott, Greenville, S. C.
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Lewis, Allan Jefferson, Haskell, Texas.
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McCrillis, Harold L., South Union and Park Ave., Des Moines, Iowa.
Maas, Edward E., Columbia, Miss.
Mack, Joseph Peter, Starke, Fla.
Martin, Capt. Arthur Dunlap, 1817 W. Pershing Road, Chicago, Ill.
Maust, Clark Jay, Red Lion, Pa.
Mericle, Robert B., Blue Earth, Minn.

Merrill, Winslow Eaton, 84 Burlington Ave., Wilmington, Mass.
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Muffly, James Albert, Watsontown, Pa.
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Murty, Byron C., Parkston, S. Dak.
Nevin, Lt. Stanley M., c/o 2nd Medical Regiment, Fort Sam Houston, Texas
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Nordstrom, Harold, Wausa, Nebr.
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Nugent, Thomas F., 210 E. Grant St., New Castle, Pa.
O'Malley, Gerald F., 93 Summit St., Clinton, Mass.
Owen, Edward Randall, Lawton, Pa.
Patton, L. E., Sidney, Mont.
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Pennell, Benjamin Nixon, 277 Williams St., New London, Conn.
Peterson, Ellis, 1914 L St., Sacramento, Calif.
Ragan, George Washington, 1308 Monroe St., Wichita Falls, Texas.
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Rawley, Walter F., Box 33, Palestine, Texas.
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Riedel, Capt. Philip H., U. S. Army, Fort Jay, N. Y.
Rynkiewicz, Joseph Bernard, New Bloomfield, Pa.
Self, Richard A., Route 8, Box 675, Dallas, Texas.
Severn, S. S., Sequin, Texas.
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Umberger, Gail M., Harveyville, Kans.
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Weber, Ralph E., Coleridge, Nebr.
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Whytock, Joseph S., 3467 La Crescenta Blvd., Verdugo City, Calif.
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Wingate, James M., Waynesboro, Miss.
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The amount which should accompany an application this month is $7.08, which covers membership fee and dues to January 1, 1930, including subscription to the JOURNAL.

Detroit has increased in population 100 per cent every ten years for the last 100 years.

John Andrew Ellens and Floyd W. Young, senior veterinary students at Michigan State College, were honored during the spring term by election to the honorary scientific fraternity, Phi Kappa Phi.

COMING VETERINARY MEETINGS

Ontario Veterinary College Veterinary Conference for Practitioners. Ontario Veterinary College, Guelph, Ont. July 29-August 3, 1929. Dr. C. D. McGilvray, Dean, Ontario Veterinary College, Guelph, Ont.

Hudson Valley Veterinary Medical Society. Kenozia Lake, Ulster County, N. Y. August 6, 1929. Dr. J. G. Wills, Secretary, 122 State St., Albany, N. Y.

Illmo Veterinary Medical Association. Annual Picnic, Marissa Country Club, Marissa, Ill. August 7, 1929. Dr. J. T. Brown, Secretary, Belleville, Ill.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. August 7, 1929. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

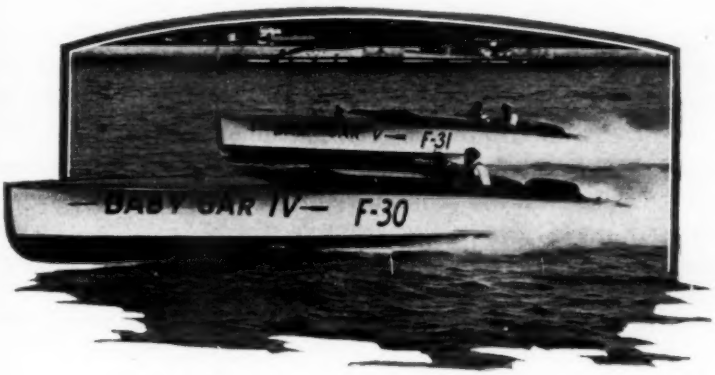
Connecticut Veterinary Medical Association. Norwich, Conn. August 7, 1929. Dr. E. H. Patchen, Secretary, Milford, Conn.

Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. August 13, 1929. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

American Veterinary Medical Association. Book-Cadillac Hotel, Detroit, Mich. August 13-16, 1929. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. August 21, 1929. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

The area of Detroit is slightly over 139 square miles.



Speedboat races on the Detroit River

THE BREEDING EFFICIENCY OF A HERD OF COWS NEGATIVE TO THE AGGLUTINATION TEST FOR ABORTION*

By HERBERT LOTHE, *Waukesha, Wis.*

On October 12, 1923, the superintendent of a herd of cows consulted me regarding the possibility of controlling abortions in his herd. He stated that the herd had been remarkably free from abortions until very recently. Just subsequent to the introduction of a number of cows from various sources during the spring of 1923 and particularly since the introduction of eleven cows on May 30, abortions had occurred in alarming numbers, both in the group recently introduced as well as in the original herd.

The history showed that the herd was largely a raised one rather than an assembled one. The herd consisted originally of grade cows but a few pure-breds had been added from time to time in 1910, 1912, 1914 and 1917, and the female offspring raised for breeding purposes until, in the spring of 1923, all grade cows were sold and enough pure-breds purchased to bring the herd to the desired size. It seemed that it was shortly after this transaction that abortions began to occur.

The superintendent was advised that every animal of breeding age in the herd should have a sample of her blood tested by the agglutination test for abortion and what plans were formulated for control would depend upon the number of animals that reacted positively to that test.

Accordingly the test was made on November 30, 1923, of the blood of 136 head, which revealed twenty-one positive reactors. Inasmuch as the number was not large (21) and as there were four cow-barns in use that were separated by distances of from one-quarter to one mile, an isolation program was decided upon. It entailed no more expense or inconvenience to have all blood reactors housed in one barn than to have them scattered throughout the herd, a few in each barn.

The following is quoted from a letter to the superintendent in December, 1923, to give an idea of the plan of procedure:

> Apply the blood test to all animals in the herd of breeding age.
> Isolate all animals giving a positive reaction to the test regardless of whether they have aborted or not; maintain these animals as a separate

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

unit or herd, allowing them no contact with the rest of the herd; provide a separate barn, paddocks, pastures and attendants.

Apply the blood test to the non-reactor group at 90-day intervals and remove the reactors to the isolation unit as they are revealed.

Isolate immediately any aborters that may appear in the non-reacting unit and apply the blood test to them.

Remove calves from the isolation unit to the calf-barn but keep them away from negative cows until some time after weaning from milk.

Use separate bulls for each group of cows, reactors and non-reactors.

This, briefly, was our plan of procedure. Allow me to quote the following from this same letter:

It should be borne in mind that abortions do at times occur from causes other than *B. abortus* Bang, in which case the cow does not react to the blood test.

The twenty-one reactors, nine of which had been recently purchased, were removed to a barn about one-half mile from the nearest barn and maintained as a separate herd with its own attendants and herd sire. Because of difficulties of a financial nature between the superintendent and those in control, the non-reacting group was not retested until ten months after the initial test. By this time abortions had begun to appear in this group although the reacting group had been maintained strictly separate from all other cows during the interval and some aborters had even been removed to the isolation unit during the period. A second test was then run, which revealed eighteen new reactors, some of which had aborted.

The eighteen new reactors added another difficulty to our program, for our isolation barn was not now large enough to hold all out blood reactors and no other building was available. To cope with this situation it was decided to place all reactors possible in the isolation barn and the excess reactors were placed in the barn that showed the largest number of reactors on the last test and remove as many non-reactors as possible from this barn to other clean barns. This arrangement left the status of our various barns as follows: one containing reactors only, two containing non-reactors and one containing reactors and non-reactors.

Subsequent test of the herd revealed from one to ten new reactors at each test, as will be noted by referring to table I. The new reactors appeared almost exclusively in the barn where reactors and non-reactors were housed together. In December of 1925, a tuberculin test of the herd showed a large number of our blood reactors to be tuberculous. (One purchased cow broke down with tuberculosis after an abortion and infected a number of her stablemates that were also blood reactors like herself.) The slaughter of the tuberculin reactors made it possible to get

TABLE I.—*Appearance of reaction in the herd*

DATE OF TEST	ANIMALS TESTED	REACTED		TOTAL
		PREVIOUSLY	FOR FIRST TIME	
11/30/23	136	0	21	21
9/23/24	134	7	18	25
12/26/24	97	5	10	15
3/7/25	77	0	1	1
6/10/25	84	0	1	1
11/13/25	162	26	10	36
11/27/26	163	19	0	19
12/16/27	132	10	0	10
Total reacted 61				

all blood reactors into our isolation barn and you will note by table I that no new blood reactors appeared from this time on.

Let us now look at the behavior of the two groups of cows, the reactors and non-reactors to the blood test, as to their breeding history. Table II gives a summary of what the two groups did at

TABLE II.—*Summary of behavior of reactors and non-reactors*

	TOTAL	ABORTED		STILL BIRTHS		PREMATURE BIRTHS		TOTAL COWS THAT FRESHENED PATHOLOGICALLY	
		No.	%	No.	%	No.	%	No.	%
Reactors*	60	20	33.3	5	8.3	7	11.6	32	53.3
Non-reactors	209	29	13.8	12	5.7	4	1.9	45	20.9
Number tested	269								

*An animal is considered a reactor when agglutination is complete in the .02 tube or higher; in other words, complete agglutination in a dilution of 1-50 or higher is considered positive.

Numbers in table indicate cows in every instance and not abortions, premature births, etc. E. g., if a cow aborted more than once, it would appear only as one in the table.

the termination of pregnancy. You will note that 53 per cent of the reactors freshened pathologically at some time during our observations, as against 20.9 per cent of the cows in the non-reacting group, a decided difference in favor of the non-reacting group.

The presence or absence of abortions alone does not constitute a true measure of a herd's breeding ability, however, for a group of barren cows would show by such a standard of measurement to be more efficient than a group that had conceived and aborted which, of course, is absurd. The ideal breeding efficiency is one calf per cow every twelve months after the animal has attained the age of fifteen months. (Breeding at fifteen months allows the heifer to freshen at two years of age.) A system of

measuring the breeding efficiency of a herd based upon this standard has been devised by Dr. W. L. Williams and described in *The Cornell Veterinarian* of October, 1919, pp. 204-213. Service and calving records of each cow in the herd we are considering had been carefully kept, so that a study of its breeding efficiency according to the above-mentioned standard and system could be made. The tabulations in table III are the result of such a study.

While a glance at table III reveals that in general the breeding efficiency of the non-reactor group is slightly higher than that of the reactor group, the table also shows a gradual decline in

TABLE III.—*Reproductive efficiency of herd*

YEAR	REACTORS (%)	NON-REACTORS (%)	
1921	54.5	93.7	
1922	71.4	70.5	
1923	72.7	85.1	Blood testing in progress this year, and following years.
1924	54.2	64.5	
1925	49.3	47.4	
1926	47.2	62.5	
1927	32.0	63.1	

efficiency of both groups from 1921 to 1927. From November 1923 the blood testing program was in operation, but, in spite of it, the efficiency has steadily declined. Evidently causes have been operative that the blood test is unable to eliminate. Table II also reveals that 29 (13.8 per cent) of our cows negative to the blood test have aborted at some time during the period of observation. By negative cows I mean those that have never reacted to the blood test either before or after the act of abortion, as shown by repeated blood tests at frequent intervals. Table IV gives the agglutination titer, dates of tests and breeding histories of this group of cows. Table V gives the same data for the reacting group.

If we accept a reaction to the agglutination test (agglutination in dilution of 1:50 (.02 cc) was considered a reaction in handling this herd) as a definite indicator of *B. abortus* Bang infection, we are forced to one of two conclusions regarding the negative abortions and declining breeding efficiency: either our test was not an accurate indicator of this infection and allowed 13.8 per cent of infected cows to "get by" or *B. abortus* Bang was not responsible.

To throw some light upon whether *B. abortus* was present, a number of fetuses from negative cows were sent to the Depart-

TABLE IV—*Cows, negative to blood test, that calved abnormally**

COW	DATES OF TESTS	BREEDING HISTORY
6	11-30-23 9-23-24	8-8-21 (H). 8-26-22 (B). 11-7-23 (H). 8-4-24 (Ab. 6 mos.). 10-8-25 (H). Died calving, 10-8-25
7	11-30-23 4-23-24 12-26-24 3- 7-25 11-13-25	8-26-21 (H). 10-2-22 (B). 10-9-22 (B). 1-14-25 (Ab. 4 mos.). Bred 7 times for last conception of 1-14-25. Slaughtered (3 mos. preg.), 12-15-25. Reactor to tuberculin
13	9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26	5-18-25 (B). 3-17-26 (Ab.). Slaughtered, 9-27-27 (barren and old age)
18	11-30-23† 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25	5-12-24 (B). 6-13-25 (Ab. 5 mos.). Treated for sterility in 1924 (metritis, left salpingitis). Slaughtered, 2-23-26 (barren, salpingitis)
32	11-30-23	12-18-18 (B). 11-4-19 (H). 11-24-20 (H). 9-6-22 (H). 1-5-23 (H). 4-14-24 (Ab.). Slaughtered, 4-30-24 (old age)
46	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 4-14-26 11-27-26 12-16-27	4-16-19 (B). 5-1-20 (H). 4-12-21 (B). 4-5-22 (H). 3-24-23 (B). 5-8-24 (H). 4-16-25 (Ab. 6 mos.). 12-9-26 (H). 6-20-28 (B)
47	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26 12-16-27 4-20-28	10-10-20 (B). 10-13-21 (H). 12-3-22 (B). 11-29-23 (B). 2-26-25 (H). 1-24-26 (Ab. 6 mos.). 4-12-27 (B). 5-2-28 (H)
54	11-30-23	3-22-17 (B). 3-17-19 (H). 4-30-20 (H). 4-31-21 (H). 5-5-24 (Ab.). Died 5-12-25 (septic metritis)

*Negative in dilutions of .02, .01, .005 and .002.
†Blood at this test gave a questionable reaction in the .02 dilution.

TABLE IV—*Cows, negative to blood test, that calved abnormally—Continued*

Cow	Dates of Tests	Breeding History
56	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26 12-16-27 4-20-28	10-23-16 (B). 7-14-17 (H). 5-6-21 (B). 6-27-22 (B). 12-14-23 (B). 6-13-24 (B). 6-7-25 (H). 7-6-26 (H). 8-10-27 (H). 5-11-28 (Ab. 5 mos.)
213	11-30-23 9-23-24 12-26-24 3- 7-25 11-13-25 11-27-26 12-16-27	9-12-22 (H). 11-28-23 (H). 1-28-25 (Ab. Fetus not found). 12-6-26 (H). Due 8-13-28
217	11-30-23 9-23-24 12-26-24 3- 7-25	10-15-22 (B). 10-8-23 (B). 8-26-24 (B. 11 days early). 2-23-25 (Ab.). Slaughtered, 8-13-25 (barren)
218	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26 12-16-27	10-26-22 (B). 12-16-23 (H). 3-28-25 (B). 5-23-26 (H. Still-birth, 22 days premature). 5-25-27 (H). Due 11-18-28
233	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25	2-27-23 (H). 2-8-24 (H). 2-3-25 (H). 10-16-25 (Ab.). Slaughtered, 5-17-26 (reactor to tuberculin and 7 mos. preg.)
226	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26	8-15-21 (H). 3-10-23 (H). 6-22-24 (H). 6-26-25 (Ab. 6 mos.). Slaughtered, 1-19-27 (barren)
235	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26 12-16-27 4-20-28	11-11-23 (Still-birth). 1-8-25 (Still-birth, 8 days premature). 2-4-26 (H). 2-3-27 (B). 5-26-28 (H)

TABLE IV—*Cows, negative to blood test, that calved abnormally—Continued*

Cow	Dates of Tests	Breeding History
236	11-30-23	10-10-23 (H). 8-10-24 (Ab.). Died 8-17-24 (septic metritis)
246	11-30-23	10-19-23 (Ab.). Slaughtered, 1-25-24 (unprofitable)
247	11-30-27 9-23-24 11-13-25 11-27-26 12-16-27	11-26-23 (H. 18 days early). 9-20-24 (Ab. 5 mos.). 11-5-25 (B). 11-29-26 (H). Slaughtered, 1-7-27 (barren). 7-3-28 (H).
252	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26	5-2-24 (Still-birth). 11-17-25 (Still-birth, full time). 1-23-27 (B). Died 1-27-27 (7 mos. preg.)
255	11-30-23 9-23-24 12-26-24 3- 7-25 11-13-25	3-22-24 (B). 4-9-25 (Ab. 5 mos.). Slaughtered, 12-15-25 (reactor to tuberculin and not preg.)
267	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26	10-28-24 (H. 24 days early). 12-25-25 (Ab. 6 mos.). Slaughtered, 4-2-27 (barren)
269	11-30-23 9-23-24 11-13-25 11-27-26 12-16-27	7-30-24 (Ab. 4½ mos.). 10-17-25 (B). 5-17-27 (B). (not preg. now)
272	11-30-23 12-26-24 3- 7-25 6-10-25 11-13-25 11-27-26 12-16-27	11-17-24 (B). 11-18-25 (H. 1 mo. early, retained afterbirth). 1-16-27 (H). (Five months pregnant now)
278	11-30-23 9-23-24 12-26-24 3- 7-25 6-10-25 11-13-25	Sept. 1924 (Ab. Unseen in pasture). 8-22-25 (H). Slaughtered, 12-15-25 (reactor to tuberculin)

TABLE IV—*Cows, negative to blood test, that calved abnormally—Continued*

Cow	Dates of Tests	Breeding History
281	11-30-23 9-23-24 12-26-24 6-10-25 11-13-25 11-27-26 12-16-27	3-8-25 (B. Still-birth). 1-11-26 (Ab. 3 mos.). 4-5-27 (B). 4-20-28 (H)
287	9-23-24 11-13-25	11-5-24 (Ab. 6 mos.). Slaughtered, 5-17-26 (reactor to tuberculin and barren)
288	11-30-23 9-23-24 12-23-24 3- 7-25 6-10-25 11-13-25 4-14-26 11-27-26 12-16-27	11-21-24 (H). 11-22-25 (Still-birth). 2-1-26 (H. and H.) 4-23-28 (H)
297	12-26-24 3- 7-25 6-10-25 11-13-25	8-5-25 (Ab. 5 mos.). Slaughtered, 12-15-25 (reactor to tuberculin and not bred since fresh last time)
299	9-23-24 1-21-25 6-10-25 11-13-25 11-27-26 12-16-27 4-20-28	7-19-26 (H. Still-birth). 9-5-27 (B)
311	6-10-25 11-13-25 11-27-26 12-16-27	4-20-26 (B. Still-birth). 4-28-27 (H). 3-4-28 (Ab. 2 mos.)
324	11-13-25 11-27-26 12-16-27	8-9-26 (B. Still-birth). (Cow very sick from metritis)
325	11-13-25 11-27-26 12-16-27 4-20-28	10-29-27 (B. Still-birth)
326	11-13-25 11-27-26 12-16-27	3-25-27 (B). Died during delivery (dystocia). 6-12-28 (B)
327	11-13-25 11-27-26 12-16-27	10-16-27 Still-birth). Cow sold 11-16-27. Open

TABLE IV—*Cows, negative to blood test, that calved abnormally—Continued*

COW	DATES OF TESTS	BREEDING HISTORY
330	11-13-25 12-16-27	12-29-26 (Still-birth)
335	1-21-25 11-13-25 11-27-26 12-16-27	8-27-27 (Still-birth). 8-18-27 (Ab. 4 mos.). 2-18-28 (Ab. 3 mos.)
346	11-27-26 5- 3-27 12-16-27	4-25-27 (Ab. 3 mos.). (Seven months pregnant now)
401	11-30-23	3-14-23 (Ab. 7 mos.). 8-21-24 (B). Cow died 8-24-24 (septic metritis)
448	11-27-26 12-16-27	11-17-26 (H). 12-7-27 (H). Cow died 12-9-27 (septic metritis)
3	11-30-23 9-23-24	1-10-22 (B). 1-9-23 (H). 9-18-24 (Ab. 4 mos.). Slaughtered, 7-9-25 (barren)
21	9-23-24 12- 6-24 3- 7-25 6-10-25 11-13-25 4-14-26 11-27-26 12-16-27	2-23-25 (B and B). 6-21-26 (B and B). 6-18-27 (Ab.)
81	11-30-23 9-23-24	10-14-23 (B). 8-19-24 (Ab.). Slaughtered, 9-13-24 (unprofitable)
86	11-30-23 9-23-24 11-13-25	5-31-23 (H). 2-1-24 (Ab. 8 mos.). 3-4-25 (B). Slaughtered, 12-15-25 (reactor to tuberculin and 7 mos. preg.)
300	9-23-24 1-21-25 6-10-25 11-13-25 11-27-26 12-16-27 4-20-28	7-12-26 (B). 8-20-27 (B). 4-1-28 (Ab. 5 mos.)
329	1-21-25 11-13-25 11-27-26 12-16-27	6-11-27 (B). 12-25-27 (Ab. 3 mos.)

TABLE V—*Reactors to blood test*

Cow	Tests: Agglutination Titer .02	.01	.005	.002	Dates	Breeding History
2	—	—	—	—	11-30-23	1-2-22 (B). 1-2-23 (B). 2-2-24 (H).
	—	—	—	—	9-23-24	8-3-25 (H). Treated for sterility
	+	+	+	+	12-26-24	1925. 7-27-26 (H). Slaughtered,
	+	+	+	—	11-13-25	12-2-26 (barren)
10	—	—	—	—	11-30-23	4-15-22 (B) 4-14-23 (B). 6-9-24 (H).
	—	—	—	—	9-23-24	6-23-25 (H). 8-25-26 (B). Slaugh-
	—	—	—	—	12-26-24	tered, 6-27-28 (barren)
	+	+	—	—	11-13-25	
	+	+	—	—	11-27-26	
	+	+	+	+	12-16-27	
16	—	—	—	—	12-26-24	2-18-25 (Calf died infected navel).
	—	—	—	—	3- 7-25	8-19-25 (Ab. 5 mos.). Slaughtered,
	—	—	—	—	6-10-25	7-3-27 (barren)
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	
19	—	—	—	—	9-23-24	2-19-24 (before purchase). 4-14-25
	—	—	—	—	12-26-24	(H). 11-7-25 (Ab.). Slaughtered,
	—	—	—	—	3- 7-25	11-30-25 (unprofitable)
	—	—	—	—	6-10-25	
	+	+	+	—	11-13-25	
20	—	—	—	—	9-23-24	4-24-24. Treated for sterility 1925.
	+	+	?	?	12-26-24	11-11-25 (B). Slaughtered, 11-10-28
	—	—	—	—	11-13-25	(barren)
	—	—	—	—	4-14-26	
22	+	+	+	—	9-23-24	2-20-24 (B). 11-24-24 (Ab. 6 mos.).
	+	+	+	+	11-13-25	Slaughtered, 12-15-25 (reactor to tuberculin and 8 mos. preg.)
41	—	—	—	—	11-30-23	11-11-17 (H). 12-13-18 (B). 11-11-
	—	—	—	—	9-23-24	19 (B). 1-3-21 (H). 1-11-22 (H).
	+	+	—	—	12-26-24	1-23-23 (H). 4-13-24 (B). 3-2-25 (B). Slaughtered, 4-21-25 (old age, unprofitable)
44	—	—	—	—	11-30-23	12-20-17 (H). 3-20-19 (H). 3-21-20
	—	—	—	—	9-23-24	(H). 3-27-21 (H). 4-14-22 (B).4-9-
	+	+	+	—	12-26-24	23 (B). 5-10-24 (H). 5-17-25 (B).
	—	—	—	—	11-13-25	Slaughtered, 12-15-26 (old age, unprofitable)
55	+	+	+	+	11-30-23	12-20-17 (H).5-31-19 (B).7-1-20 (B).
	+	+	?	—	3-12-24	12-22-21 (B). 12-30-22 (H). 1-20-24 (B and B). Died at term. Slaughtered, 5-28-24 (unprofitable and not pregnant)

TABLE V—*Reactors to blood test—Continued*

Cow	Tests: Agglutination Titer .02	.01	.005	.002	Dates	Breeding History
60	+	+	?	—	11-30-23	2-21-14 (H). 12-7-15 (B). 4-14-1
	+	+	?	—	3-12-24	(B). 4-18-19 (B). 7-7-20 (B). 7-30- 21 (H). 7-6-22 (H). 12-28-23 (H). Slaughtered, 4-28-24 (old age, un- profitable)
66	—	—	—	—	11-30-23	5-28-24 (H). 7-8-23 (H). 8-30-24 (B).
	—	—	—	—	9-23-24	Slaughtered, 1-14-25 (unprofitable)
	+	+	+	—	12-26-24	
82	+	+	+	?	11-30-23	7-16-23 (Ab. 8 mos.). 8-19-24 (H).
	+	+	+	?	3-12-24	Slaughtered, 9-22-26 (barren)
	+	+	+	—	9-23-24	
	+	?	—	—	6-10-25	
	+	?	—	—	11-13-25	
85	+	+	+	+	11-30-23	9-8-23 (Ab.). 3-22-25 (H). 11-21-26
	+	+	+	+	3-12-24	(H). 2-6-28 (B)
	+	+	+	+	9-23-24	
	+	+	?	—	11-13-25	
87	—	—	—	—	11-30-23	12-23-23 (B). 3-2-25 (Ab. 6 mos.).
	—	—	—	—	9-23-24	Slaughtered, 12-15-25 (reactor to
	—	—	—	—	12-26-24	tuberculin and not pregnant)
	+	+	+	?	11-13-25	
88	+	?	—	—	11-30-23	5-11-24 Still-birth). (5-11-25 (B).
	?	—	—	—	3-12-24	Slaughtered, 12-15-25 (reactor to
	—	—	—	—	9-23-24	tuberculin and not pregnant)
	+	—	—	—	11-13-25	
90	+	+	+	—	11-30-23	11-15-23 (Ab.). 12-16-25 (H). 3-3-
	+	+	+	—	3-12-24	26 (H). Slaughtered, 1-3-27 (reac-
	+	—	—	—	9-23-24	tor to tuberculin and 5 mos. preg-
	+	+	+	+	11-13-25	nant)
	+	+	+	+	11-27-26	
99	+	+	?	—	11-30-23	11-26-17 (H). 5-17-19 (H). 10-12-21 (B). 9-29-23 (H). Slaughtered, 1-25-24 (unprofitable)
112	+	?	—	—	11-30-23	2-18-19 (H). 2-25-20 (B). 5-17-21 (B). 5-10-22 (H). 5-19-23 (B). 8-20-23 (Ab.). Slaughtered, 1-30-24 (barren)
152	+	+	+	?	11-30-23	10-6-22 (B). 9-18-25 (H). 9-14-24
	+	+	?	—	3-12-24	(H. 14 days early). Calf lived.
	+	—	—	—	9-23-24	10-31-25 (H). Slaughtered, 12-15-25
	+	—	—	—	11-13-25	(reactor to tuberculin)

TABLE V—*Reactors to blood test—Continued*

Cow	Tests: Agglutination Titer .02	.01	.005	.002	Dates	Breeding History
199	—	—	—	—	11-30-23	8-26-22 (B). 9-20-23 (B). 8-26-24
	—	—	—	—	9-23-24	(B). 11-2-25 (Ab. twins). 1-27-27
	—	—	—	—	3- 7-25	(B). Slaughtered, 5-3-28 (barren)
	+	+	+	+	11-13-25	
202	—	—	—	—	11-30-23	10-20-22 (H). 2-5-23 (H). 1-21-24
	+	+	+	+	9-23-24	(H). 10-16-25 (H). Slaughtered,
	+	+	+	+	12-26-24	1-19-27 (barren)
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	
204	—	—	—	—	11-30-23	2-7-22 (H). 1-26-23 (B and B).
	+	+	—	—	9-23-24	1-13-24 (H. 14 days early). Lived. Slaughtered, 7-22-25 (barren)
206	—	—	—	—	11-30-23	1-11-21 (H). 3-6-22 (H). 4-6-24
	+	+	+	+	9-23-24	(Ab. 6 mos.). Slaughtered, 12-5-25
	+	+	+	+	11-13-25	(reactor to tuberculin and one month pregnant)
214	—	—	—	—	11-30-23	9-9-22 (H). 9-11-23 (H). 8-3-24
	+	+	+	+	9-23-24	(Ab. 7 mos.). 10-19-25 (H). 2-9-27
	+	+	+	+	11-13-25	(B. Still-birth). Slaughtered, 12-23-27 (reactor to tuberculin)
216	—	—	—	—	11-30-23	10-17-22 (B). 9-11-23 (Ab.). 7-24-24
	—	—	—	—	3-12-24	(Ab.). Slaughtered, 12-15-25 (re-
	+	+	+	+	9-23-24	actor to tuberculin and three
	+	+	+	?	11-13-25	months pregnant)
219	+	+	+	?	11-30-23	10-24-22 (H). 9-2-23 (Ab.). 8-30-24
	+	+	+	+	3-12-24	(B). 9-27-25 (B). Slaughtered,
	+	+	+	+	9-23-24	12-15-25 (reactor to tuberculin)
	+	+	+	+	11-13-25	
221	—	—	—	—	11-30-23	2-2-23 (H). 1-21-24 (B). 4-27-25 (B).
	+	—	—	—	9-23-24	4-5-26 (B) 12-25-27 (B)
	+	+	+	+	12-26-24	
	+	+	+	?	11-13-25	
	+	+	+	?	11-27-26	
	+	+	+	+	12-16-27	
222	—	—	—	—	11-30-23	2-15-23 (B). 1-28-24 (H). 8-9-25
	+	+	+	—	9-23-24	(Ab. 5 mos.). Slaughtered, 10-21-26
	+	+	+	+	12-26-24	(barren)
	Tube broken				11-13-25	
224	+	?	—	—	11-30-25	3-28-23 (B). 2-14-25 (H). Slaugh-
	?	—	—	—	3-12-24	tered, 12-15-25 (reactor to tuber-
	—	—	—	—	9-23-24	culin and three months pregnant)
	+	+	+	—	11-13-25	

TABLE V—*Reactors to blood test—Continued*

Cow	Tests				Dates	Breeding History
	Agglutination Titer					
	.02	.01	.005	.002		
228	—	—	—	—	11-30-23	3-28-23 (B). 3-20-24 (B). Slaughtered, 2-7-25 (unprofitable)
	+	+	+	+	9-23-24	
	+	+	+	+	12-26-24	
229	—	—	—	—	11-30-23	4-22-23 (H). 4-13-24 (B). 10-20-25 (H). Slaughtered, 12-14-26 (barren)
	—	—	—	—	9-23-24	
	+	+	—	—	12-26-24	
	—	—	—	—	11-13-25	
230	—	—	—	—	11-30-23	4-23-24 (H). 6-7-24 (B. Still-birth). 7-28-25 (B). Slaughtered, 4-27-27 (barren-bilateral salpingitis)
	—	—	—	—	9-23-24	
	—	—	—	—	12-26-24	
	—	—	—	—	3- 7-25	
	—	—	—	—	6-10-25	
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	
231	—	—	—	—	11-30-23	4-29-23 (B). 5-13-24 (H). 5-11-25 (B). 8-24-26 (B). 12-5-27 (B)
	—	—	—	—	9-23-24	
	—	—	—	—	12-26-24	
	—	—	—	—	3- 7-25	
	+	—	—	—	6-10-25	
	+	+	+	+	4-14-26	
	+	+	+	+	11-27-26	
	+	+	+	+	12-16-27	
233	+	+	+	+	11-30-23	Slaughtered, 7-14-24 (barren). No breeding history prior to 5-24-23
	+	+	+	+	3-12-24	
234	+	?	—	—	11-30-23	8-23-24 (H). 7-14-24 (B). 2-13-26 (H). 10-24-27 (H). Slaughtered, 5-3-28 (unprofitable)
	?	—	—	—	3-12-24	
	—	—	—	—	9-23-24	
	—	—	—	—	11-13-25	
	—	—	—	—	11-27-26	
	—	—	—	—	12- 6-27	
238	—	—	—	—	11-30-23	12-17-23 (B). 7-4-25 (H). Slaughtered, 1-19-27 (unprofitable—udder infected)
	+	+	+	+	9-23-24	
	+	+	+	+	12-26-24	
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	
240	—	—	—	—	11-30-23	10-8-23 (H). 8-26-24 (Ab. 8 mos.). Calf lived. 11-25-25 (B). Slaughtered, 12-15-25 (reactor to tuberculin)
	—	—	—	—	9-23-24	
	+	—	—	–	11-13-25	
241	—	—	—	—	11-30-23	11-14-23 (B). 3-3-25 (H). Treated for sterility 1924. Catarrhal endometritis. 3-7-26 (B). 5-4-27 (H)
	+	+	+	+	9-23-24	
	+	+	+	+	12-26-24	
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	
	+	+	+	+	12-16-27	

TABLE V—*Reactors to blood test—Continued*

Cow	Tests					Breeding History
	Agglutination Titer				Dates	
	.02	.01	.005	.002		
248	—	—	—	—	11-30-23	4-11-24 (B). 3-18-25 (Ab. 8 mos.). 6-5-27 (H)
	+	+	+	+	12-26-24	
	+	+	+	+	11-13-25	
	+	+	+	+	12-16-27	
253	—	—	—	—	11-30-23	1-28-24 (H). 3-5-25 (H). 3-7-26 (B). 5-28-27 (H). Cow died (septic metritis)
	—	—	—	—	9-23-24	
	+	+	+	—	12-26-24	
	?	—	—	—	11-13-25	
	+	?	—	—	4-14-26	
	—	—	—	—	11-27-26	
	+	+	?	—	12-16-27	
254	—	—	—	—	11-30-23	3-31-24 (B). 3-25-25 (B). Slaughtered, 12-16-25 (reactor to tuberculin)
	—	—	—	—	9-23-24	
	+	+	+	+	11-13-25	
256	—	—	—	—	11-30-23	4-14-24 (H. 26 days early). 6-25-26 (B. Still-birth). 4-27-28 (H).
	+	+	+	—	9-23-24	
	+	+	+	—	11-13-25	
	+	?	—	—	11-27-26	
	+	+	+	?	12-16-27	
257	—	?	—	—	11-30-23	6-28-24 (H. 16 days early). Slaughtered, 5-22-25 (barren)
	+	+	+	+	9-23-24	
259	+	?	—	—	11-30-23	3-21-24 (Ab.). Slaughtered, 10-25-24 (reactor to tuberculin and not pregnant)
	+	+	+	+	9-23-24	
260	+	+	+	+	11-30-23	12-22-23 (Ab.). Slaughtered, 4-23-24 (reactor to tuberculin)
	+	+	+	+	3-12-24	
261	—	—	—	—	11-30-23	5-4-24 (H). Slaughtered, 9-23-25 (reactor to tuberculin and 8 months pregnant)
	+	—	—	—	9-23-24	
	+	+	+	+	12-26-24	
263	—	—	—	—	11-30-23	3-10-24 (H). 3-19-25 (H). 4-4-26 (B). 1-15-28 (H)
	+	+	+	—	9-23-24	
	+	+	+	+	12-26-24	
	+	+	?	—	11-13-25	
	+	+	+	—	11-27-26	
	+	+	+	+	12-16-27	
265	—	—	—	—	11-30-23	3-17-24 (B). 8-22-25 (H. 1 mo. early). Died (premature birth). Slaughtered, 7-20-27 (barren)
	+	+	+	—	9-23-24	
	+	+	+	+	12-26-24	
	+	+	+	+	11-13-25	
	+	+	+	+	11-27-26	

TABLE V—*Reactors to blood test—Continued*

Cow	Tests: Agglutination Titer .02	.01	.005	.002	Dates	Breeding History
266	—	—	—	—	11-30-23	10-28-24 (H). 10-7-25 (H). 4-13-2
	—	—	—	—	9-23-24	(B). Died, 5-19-27 (cardiac tumor
	+	—	—	—	12-26-24	
	+	+	+	—	11-13-25	
	+	+	+	+	11-27-26	
270	—	—	—	—	11-30-23	12-11-24 (H). 11-9-25 (B). Slaugh-
	—	—	—	—	9-23-24	tered, 1-19-27 (barren, nympho-
	—	—	—	—	12-26-24	mania cystic ovaries)
	—	—	—	—	3- 7-25	
	+	+	—	—	6-10-25	
	+	+	+	+	11-13-25	
					12-27-26	
271	+	+	+	?	11-30-23	10-7-24 (H). Slaughtered, 12-15-25
	+	+	+	+	3-12-24	(reactor to tuberculin)
	+	+	+	+	9-23-24	
	+	+	+	+	11-13-25	
284	—	—	—	—	9-23-24	4-26-25 (H). 4-16-26 (H). 7-8-27 (B)
	+	+	+	—	12-26-24	
	+	+	+	—	11-13-25	
	+	+	?	—	11-27-26	
	+	+	+	+	12-16-27	
316	—	—	—	—	9-23-24	9-21-23 (H). 9-19-24 (H. 10 days
	+	+	+	+	11-13-25	early). Slaughtered, 12-15-25 (re-
						actor to tuberculin and six months
317	—	—	—	—		pregnant)
	—	—	—	—	9-23-24	
	+	?	—	—	12-26-24	1-4-24 (H). Died. 6-18-25 (H).
	—	—	—	—	3- 7-25	4-2-27 (H). 7-1-28 (B)
	—	—	—	—	6-10-25	
	—	—	—	—	11-13-25	
	—	—	—	—	11-27-26	
					12-16-27	
400	—	—	—	—	11-30-23	10-7-24 (Ab.). 12-7-25 (Ab.).
	—	—	—	—	3- 7-25	Slaughtered, 12-15-25 (reactor to
	+	+	+	—	11-13-25	tuberculin)
501	+	+	?	—	11-30-23	No history prior to 1-25-23. Barren,
	+	+	+	—	3-12-24	returned to seller 6-1-25 (bilateral
503	+	+	+	?		salpingitis)
	?	—	—	—	11-30-23	12-22-23 (B). Slaughtered, 1-10-24
505	?	—	—	—	3-12-24	(unprofitable)
	+	+	+	—	11-30-23	1-10-24 (B. 1 mo. premature). Died
	+	+	?	—	2- 1-24	(goitre). (Slaughtered, 8-24-24 bar-
					3-12-24	ren)

Two cows' records lost.

ment of Veterinary Science of the University of Wisconsin for cultural examination for *B. abortus*. All such fetuses failed to reveal this germ, while fetuses from positive cows forwarded at different times showed this organism present. A number of virgin heifers were allowed to associate in intimate contact with cows that belonged to the negative abortion group, bred and continued their association to see if anything happened. None of the heifers became reactors to the test nor did any of them abort. It did not seem that the test was inaccurate and we are forced to the conclusion that causes other than *B. abortus* were responsible for the lowered breeding efficiency and abortions in the negative group.

During 1925, we noticed that a larger percentage of cows that had conceived to one bull aborted than those that had conceived to other bulls used and that these cows did not react to the blood test. This bull had been purchased in 1923, to be used upon the daughters of the senior herd sire, but other cows also were bred to him. The only thing noted clinically was slowness of service and a more or less severe balanitis that would respond to local antiseptic treatment, only to reappear in a short time. A tabulation of his services and conceptions is shown in table VI under bull NSBOC. You will note that his percentage of conceptions is low and the pathological births run from 30 to 50 per cent. Further study of the breeding efficiency of the bulls used revealed others of lowered efficiency.

The bull PMF (table VI) was purchased to replace the bull NSBOC, slaughtered because of being a breeding failure. This bull was purchased from the same person as NSBOC and had been used for light service in this man's herd before sale. Both of these bulls show up badly as to percentage of conceptions and pathologic births from the very first introduction to the herd. The bull KCHS shows a much higher breeding efficiency than either of the two above mentioned, but the influence of the other two is made apparent from 1923 on. This was probably due to the fact that he was frequently used upon cows that had failed to conceive to services by the other two, many of which were hard to "get with calf"; in fact, a large number of virgin heifers (17) became helplessly barren after repeated services to these bulls, due to clinically recognizable salpingitis (oviducts as large in diameter as a lead pencil plainly palpable per rectum) and sent to slaughter in 1924 to 1926.

The record of the bull MOH is interesting in that it shows a gradual decline of his breeding powers. His breeding alibity was not the best from the beginning of his career, but we did not suspect him, for breeding difficulties were not unusual in cows that has been bred to NSBOC and PMF and many served by him had been so bred. In 1927 he exchanged barns with the bull KCHS and was given heavier service than previously, which is reflected in his lower breeding efficiency as shown in table VI*. In the winter of 1927-28, a considerable number of cows bred to him failed to show estrum following service and were found to be carrying no calves, but instead their uteri contained from one to several quarts of white fluid; cows bred to other bulls showed no such fluid collections. It should be stated that some cows bred at this time by MOH conceived, but quite a large percentage were in the above condition; many of the calves born the result of services by MOH showed an intense yellowish discoloration of the body, while calves born to services by the bull KCHS born in the same barn were nice and clean in appearance. What significance this discoloration has I do not know, but it occurred so often that the attendants noted it in most of the calves sired by this bull. MOH was retired from service (slaughtered) in 1928.

The graph in figure 1 gives the combined services and conceptions of the three bulls NSBOC, PMF and MOH, with the percentage conceptions from 1923 to 1927. (This was obtained by adding the services and conceptions of the three and computing percentages by years.) The graph shown was obtained by platting the efficiency of the three bulls upon the same scale as the efficiency of the non-reacting group of cows. The two curves show very much the same contour and picture the influence these bulls had upon the breeding efficiency of the herd. Since the negative abortions were a part of the lowered efficiency, it would seem reasonable to assume that the bulls in question probably contributed to their occurrence also.

But you may object on the ground that the bull KCHS also sired pathologic births as shown by his breeding record. This is true, but they did not make their appearance until after the introduction of the bulls NSBOC, in 1923, and PMF, in 1925. After the disposal of NSBOC, his breeding efficiency increased somewhat, only to drop again in 1927, when he was moved to the herd bred by MOH (see figure 2). MOH was a son of KCHS,

*Table VI is not published, due to its great length and consequent lack of space. EDITOR.

hence was used upon much the same cows as KCHS for, obviously, he was not used upon the daughters of KCHS (his sisters) on account of inbreeding. Many of the pathologic births by KCHS were in cows that has been bred previously to one of the three bulls in question.

Year	1923	1924	1925	1926	1927
Services	6	94	124	138	160
Conceptions	4	31	39	43	38
Percentages	66.6	31.9	31.4	31.1	23.7

FIG. 1. Services and conceptions of three bulls with low breeding efficiency.

While the evidence presented implicating the three bulls as contributing to the lowered efficiency of KCHS is largely circumstantial, the fact still remains that the three bulls in question sired more pathologic births than other bulls used. What was the nature of the infection, if it were an infection that caused the disturbances, I am unable to state; all I know is what occurred.

You may ask, what is my reaction to the results pictured here. Do not the figures given tend to discredit the blood test?

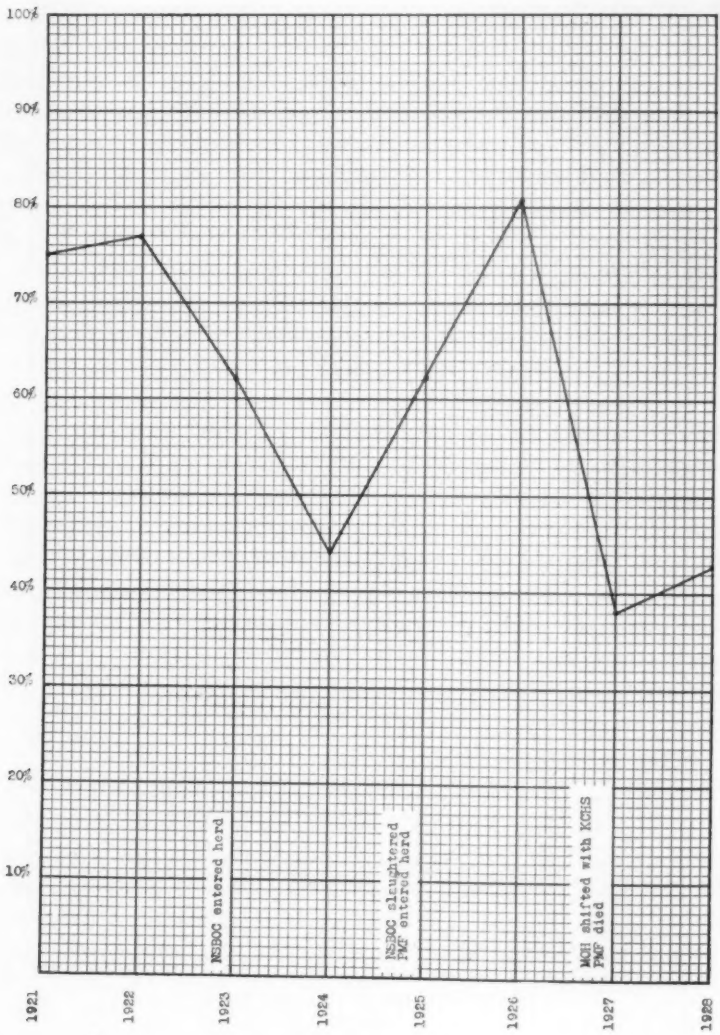


FIG. 2. Efficiency curve of bull KCHS.

Table I shows conclusively that agglutination reactors can be eliminated from the herd by testing and isolation. With removal of reactors, *B. abortus* Bang also is removed from the herd. *B. abortus* has been definitely indicated as a cause of abortion in cows, hence its elimination is a worthy and desirable accomplishment. More cows carrying abortion infection (*B. abortus* Bang) abort than those that do not and abortion is a cause of breeding inefficiency, hence blood testing and elimination of reactors should tend to increase the breeding efficiency of the herd. I have no way of knowing what would have occurred in the herd we have just been discussing, had the non-reacting unit not been protected against *B. abortus* Bang infection by testing and isolation; I rather imagine, from observations in other herds, that we would have had a more disastrous story to relate than is pictured by the tabulations above.

Blood testing did not prevent the appearance of abortions in all negative cows, in fact, a larger number of negative cows (29) aborted than positive (20). (See figure 3.) Factors other than

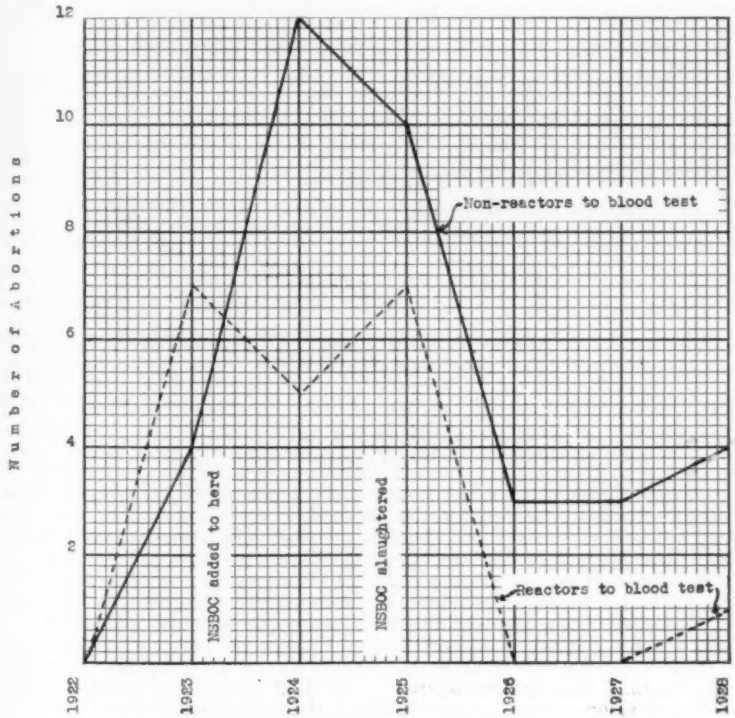


FIG. 3. Number of abortions in reactor and non-reactor groups.

B. abortus were responsible for the abortions of these cows and the blood test was unable to prevent their occurrence. The test is not a test for the act of abortion, but for antibodies in the blood that are the result of a response of the animal body to the presence in the body of *B. abortus*. Bang A large percentage of cows so infected abort, but not all abortions are so caused. *B. abortus* is *a* and not *the* cause of abortion although probably it is the greatest one cause. The results tabulated above show to me conclusively that factors other than *B. abortus* play a part in the cause of abortion, and, in individual herds, a not inconsequential part. These results show us the danger of making extravagant claims for the blood test with corresponding discouragement of the cattle-owner when he finds that some of his non-reacting cows abort. Figure 3 shows that abortions of this nature may even exceed those due to Bang infection.

I know of no effective way of preventing the introduction of genitally diseased bulls into a herd except history, and this is not usually available. Microscopic examinations of semen and clinical examinations have often been disappointing in my hands. The keeping of complete breeding records probably offers the best protection. The field is a fertile one for investigation.

I want to state in closing that I have the very highest regard for the blood test, but let us not expect too much of it. If you are dealing with *B. abortus* infection alone in a herd, your results will be encouraging, but you must be on the lookout for other disturbances to reproduction and not expect the blood test to eliminate these also. It is not a panacea for all causes of abortion and breeding irregularity. Someone has stated that in visiting a herd of sick hogs, "don't overlook hog cholera virus as a cause." The same holds for a herd of cows where abortions are occurring. Eliminate *B. abortus* Bang as a cause only after conclusive proof of its absence (blood testing) but realize that other causes of abortion may be present.

Discussion

Dr. C. H. Case: In the last table Dr. Lothe displayed, the negative cows showed twelve per cent abortion and the positive cows, I guess, seven or eight. The positive cows that showed abortion are cows you separated and put over in the other stable?

Dr. Lothe: Yes.

Dr. Case: From my experience, and that of others, eighty per cent of the cows that abort once, abort again. Did those cows abort before?

Dr. Lothe: Some of them had already aborted prior to the blood test, and some aborted subsequent to the blood test.

Dr. Case: You are considering cows that may have aborted before, probably developed some protective immunity, and will not abort again?

Dr. Lothe: Yes.

DR. CASE: Most authors will tell you that twelve to fifteen per cent of the abortions are from causes other than the Bang organism. In explaining that table, it looks as though you got more abortions in the negative cows, while really you were dealing with the cows that had aborted once, or negative cows, that won't abort again.

DR. LOTHE: The broken line (fig. 3) represents cows from 1922 on. You will remember I told you that in 1923 abortions began to appear in alarming numbers in the herd in question, and prior to that they had no abortions in the herd. This broken line will show you that in 1923 there were seven abortions in the herd. Those seven cows, as I recall it, all reacted to the blood test. Up until the starting of the blood test there had been no abortions in the herd. There had been no abortions at all for the past three or four years. Beyond that time I couldn't get any breeding data. But the abortions that appeared from here on, during 1923, 1924 and 1925, amongst our reacting group, were cows that had aborted prior to 1923, but a lot of them aborted after the blood test was made. So they were not old aborters; they were not immune.

This curve should give you a fairly good cross-section of what the abortion infection did in the herd. It also shows that your negative abortions were higher. You will notice that this bull NSBOC was purchased here, and immediately our number of negative aborters jumped up. When he was slaughtered, our curve dropped down immediately. Whether the bull NSBOC brought the infection or the disturbance into the herd, and it was transmitted to the other two bulls, I don't know. The bull PMF was purchased from the same owner as the bull NSBOC, and had been used for like service on his herd prior to the time he was purchased.

Whether this bull was the cause of the disturbance and brought it into the herd and transmitted it to these other bulls, I am sure I do not know. But all I know is that it appeared and it tended to disappear after these bulls were retired from service.

DR. CASE: Were any of these bulls ever positive?

DR. LOTHE: No.

DR. G. H. OAKES: In your classification of the pathological births, you have one that aborted; another one, still-births; and another one, premature births. I would like to have you explain that. (Table II).

DR. LOTHE: Aborted means that the animal calved and dropped a fetus that was unable to live. It wasn't viable. The still-births were those that freshened at full time, but the calves were dead. The premature births were calves that were dropped earlier than the full time of gestation, but lived. They might have been dropped at eight months and have lived.

DR. E. E. WEGNER: I would like to ask Dr. Lothe to tell us what differences he observed in the production record of these two herds, negative and positive —the milk record.

DR. LOTHE: In general, the amount of milk given by the reacting group was lower than that given by the non-reacting group. I will state however, that this bunch of reactors had to pay its way, and the herd was made to pay by vigorous culling. The minute an animal was not paying, it was sent away for slaughter.

On the whole, our reacting group has been profitable, probably not so profitable as a similar number of non-reacting cows, but we had to keep a certain number of cows in that group in order to maintain that farm. They were simply kept. Of those sixty reactors we had in 1923, I think we have thirteen left. The herd has dwindled down to about thirteen.

In 1925 we lost some twelve or fifteen by the tuberculin test. In all, I think during this period there have probably been some twelve or fifteen that have been sent away as being barren, and the rest of them have been sent away on account of being unprofitable. But there is a decided difference in the milk-production of the negative cows. I should say somewhere in the neighborhood of twenty per cent.

DR. WARD GILTNER: Mr. Chairman, I ask pardon for invading this section but I think it is only fair to warn the practitioners that this case, which is very nicely presented by Dr. Lothe, should not disturb our view of Bang's disease.

We encounter these herds where there is Bang's disease and some other cause of abortion. I don't understand that Dr. Lothe gives the cause of this other kind of abortion, nor do we find exactly what it is in these herds. It is not surprising that you get the results, that he had pictured here, in occasional herds. We reported on a similar situation in another section yesterday.

The lowered production in the Bang-disease group can be readily explained by our knowledge of the action of Bang's bacillus on the udder of these cows, in addition to the effect of an abortion on lowered production. These other causes of abortion in all probability, do not have any action directly on the udder, so that your lowered milk-production is traceable to the abortion.

I think you men should not lose sight of the value of the agglutination test in your efforts to handle Bang's disease. I am not in a position to state what percentage of herds affected with abortion will show abortion due to other causes, but I think it is very small. When you do encounter one of these herds, you should simply realize that you are dealing with Bang's disease and with some other disease, or perhaps diseases. But don't give up your faith in what you have been taught relative to handling Bang's disease.

In three groups of animals we reported on, we found that we had excellent results with our efforts in two of those groups, where we were dealing with Bang's disease pure and simple, but not where we had such a situation as Dr. Lothe has spoken about.

DR. T. A. SIGLER: Did you make any postmortem examination of these three bulls at the time they were killed?

DR. LOTHE: The only one on which I had a postmortem was bull PMF that died from pyothorax. As far as I was able to determine grossly, there was no microscopic examination made. I could detect no evidences of disease in the genital tract. I will admit, though, that the postmortem examination was somewhat superficial. No pathological, no bacteriological or histological work was done.

DR. CASE: Did you say you examined the semen of the bulls?

DR. LOTHE: Yes.

DR. CASE: Were the tails all normal?

DR. LOTHE: As far as I was able to determine, the motility was all right. In my limited knowledge of the histopathology of the semen of bulls, they looked all right. I also sent samples of the semen away to be examined by competent pathologists, and they have reported that the semen of at least one of these was normal. Nevertheless, this bull was still producing this disturbance.

The examination of semen has been rather disappointing in my hands. If you find in examining semen that your motility is nil, or that you have a lot of tailless heads, then, of course, you are justified in assuming the bull is ineffective or inefficient as a breeder. So many of them, where the clinical history has led me to believe they are not in ship-shape as far as breeding ability is concerned, I have had reported back to me from the laboratory that they are absolutely O. K. Still they will not settle cows in calf. For that reason I have rather lost faith in the microscopic examination of semen, that is, not in all cases, but my clinical observation, I believe, has the microscopic examination beaten to death. The only sad part of it is, of course, that you don't get your clinical history until after they have been used, and you have already gotten into trouble.

I might relate the instance of one bull in particular. This bull was used in this herd that I just mentioned, but he bred only four cows in this herd. These four cows happened to be cows of unusual individuality—show animals. This bull happened to be a bull of unusual individuality that also sired animals of unusual individuality. They were desirous of breeding some of the best individual cows to this bull. When I heard of it, I was sorry they had done so. I advised them to that effect, because I knew the breeding conditions of the herd in which this herd sire was being used.

On examination of three of these four cows at from 60 to 120 days after they were bred, there was revealed that the uteri contained a considerable quantity of whitish fluid that I designated as pus. They were not pregnant. On appropriate treatment they became pregnant when bred to other bulls.

This bull was later sold to some people that lived about forty miles from my home. They used him on some thirty-odd cows during the following year, with the result that only one conceived. He was therefore returned to the original seller. I later visited this farm where they had the one conception and found that, instead of the animal being pregnant, her uterus contained a large amount of pus.

After the return of the bull he was transferred to the farm of another client of mine. I advised him, "Don't use the bull until I make a microscopic examination of him to see what condition he is in." I examined some fresh material, and there apparently was sufficient motility. There was nothing I could criticize. But I also forwarded a sample to the laboratory, to be examined, giving the history of what this bull had done on the three herds I knew he had been used upon. I received a report that the bull's semen was normal, and the trouble was probably with the cows. Nevertheless, I advised these people to use this bull only upon their grade cows, and to pick out some that weren't of much value. I will state at this time that the bull was in better physical condition than he was at the time he was used upon the four cows in the herd just mentioned.

There were eighteen grade cows bred to this bull with no conceptions, with the possible exception of one. This cow was examined after she had been bred six months, and her uterus was found to contain some three or four quarts of whitish pus, the same as the rest of them.

The laboratory report on this bull was that he was absolutely O. K., and the clinical results were that he was no good at all. I can't quite reconcile those different results.

DR. W. W. ARZBERGER: I should like to ask Dr. Lothe if he practices any special sanitary precautions in either of the herds, or just the natural conditions.

DR. LOTHE: There were practically no sanitary precautions taken. The barns were of the very best dairy type. There was no attention paid to the douching of the bulls prior and subsequent to service. In fact, the animals were left pretty much to the herd conditions.

DR. J. W. CONNAWAY: Mr. Chairman, just a word on that. I wish to commend this paper which Dr. Lothe has presented, because I believe it is one of the best presentations of this subject that I have listened to, in that it presents two sides of the shield, on the opposite sides of which different groups have been siding, one group laying great emphasis on the abortion test, and another group laying great emphasis on other factors. The doctor has brought these things together and presented them in a way that shows us the great importance of both sides.

Two years ago, I had the temerity to present an illustrated lecture down at Lexington before this group. I butted in. I felt that it was time to butt in with the laboratory features. I think the practitioners all enjoyed what I presented, in standing up for the great importance of the blood test in the eradication and control of this disease.

At that time one of the chief opponents of the blood test was the chairman of the same committee or division that Dr. Giltner has mentioned. Our trouble has been that we haven't put before the group of practitioners who have to do the practical work, the information in a way that they can use it.

We are glad to hear Dr. Lothe value the importance of this test. We have always known that he valued the importance of this test as much as anyone else. He has presented the matter in a way which I hope will forever silence the objectors to the blood test as a practical measure in the control of abortion disease.

I want to relate just one instance which will show up the great importance of the bull side. Here is one thing we must look out for in breeding efficiency. There is a tendency now among the dairymen to conserve and preserve and use old bulls which have made big records, whose progeny have made big records as milk-producers. That is important, if we can get good breeding efficiency out of those bulls. So the health of the bull is one of the strong points which Dr. Lothe has brought out, and one that we must not undervalue in the least.

The case I refer to is this: A man came into our laboratory one day scared to death. He had a big herd of white-face registered cattle, pure-bred cattle. He was getting no calves. He had bought an old bull which had been overused in another herd, and was on his last legs as a breeder. He got him cheap, but he thought "Here is an opportunity for me to improve my herd." After trial he was getting no calves; had an abortion or two. He thought that the trouble was abortion disease.

I sent one of our men to his herd, but from his story I suspected the bull was the trouble and not the cows. This man whom I sent to his herd drew blood samples from all of his cows and from the bull. He examined the bull's semen and found that this bull was infertile. The blood samples were tested, and he didn't have a single reactor in his herd.

A change to a younger bull and a fertile bull cured what was apparent sterility in his herd. His cows were all right. (Applause)

Detroit has forty-two concerns engaged in the manufacture of airplanes.

AN OLD-TIMER

Dr. A. E. Howell, Homer's 90-year old practicing veterinarian, cast his first presidential vote back in 1860, when Abraham Lincoln was elected president. He is believed to be the oldest practicing veterinarian in Michigan, if not in the whole United States.

Marshall (Mich.) *Chronicle*

Detroit has approximately 400 schools.

General Motors Building, Detroit

TULAREMIA: ITS TRANSMISSION TO SHEEP BY THE WOOD TICK, DERMACENTOR ANDERSONI STILES

By R. R. PARKER, *Special Expert, United States Public Health Service, Hamilton, Montana, and*
JAMES DADE, *Chief Inspector, Idaho State Sheep Commission, Boise, Idaho*

In the spring of 1923 there were reported to the field station of the Public Health Service at Hamilton, Montana, several instances of heavy losses among sheep, that were believed due to the wood tick, *Dermacentor andersoni* Stiles. These reports were from eastern Montana and southern Idaho and were the first complaints of probable tick-caused trouble in sheep that had been received. Each report was investigated by Parker, either by personal visit to the locality concerned or by correspondence, and in one instance ticks from affected eastern Montana sheep were tested at the Hamilton laboratory. Due to the short period during which the sheep were affected and the fact that most of the reports were received after the trouble was past, no very suggestive data were secured.

At the time, it was thought likely that the losses were due to tick paralysis, which was first reported in sheep in British Columbia by Hadwen,[1] in 1914, and has since been observed there by Bruce.[2,3] The symptoms reported from eastern Montana especially were suggestive of this condition, but the death, from a then undetermined infection,* of all guinea pigs into which the ticks noted above as recovered from affected eastern Montana sheep were injected, did not seem to support this possibility. It was not until later in the season, however, after the demonstration by Parker, Spencer and Francis,[4] of the ability of *D. andersoni* to acquire and transmit tularemia, and of the occurrence of tularemia infection in over-wintered adult ticks collected in nature, that the possibility that *Bacterium tularense* might be responsible for the reported trouble was seriously considered. This likelihood seemed the greater because in each of the several regions where trouble had occurred that spring, a record had

*In view of later experience this infection was almost certainly tularemia. Ticks infected with *Bacterium tularense*, when injected intraperitoneally into guinea pigs, frequently cause death without typical lesions. These usually become typical if transfer by tissue emulsion is made to a second guinea pig. This, however, was not known at the time of the above tests.

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been secured of a coincident high mortality among jack rabbits. Dead rabbits were also reported as heavily tick-infested. It was, therefore, thought possible, as is now known to be true both for the wood tick and for the rabbit tick (*Haemaphysalis leporis-palustris* Packard), that infected over-wintered wood ticks were transmitting tularemia to rabbits and also that such infected wood ticks might be concerned in the coincident illness and mortality among sheep.

This possibility was first suggested to Dr. W. J. Butler, State Veterinary Surgeon of Montana, and later to the other author of this paper, Dr. J. S. Dade, Chief Inspector of the Idaho State Sheep Commission, and an informal arrangement was made with each for the joint investigation of future outbreaks in their respective states.

Various circumstances prevented the securing of satisfactory data from affected bands in either state until the spring of 1928, when the authors were able to study the severely affected Blackwell band near Boise. The results of this study have indicated that, as far as southern Idaho is concerned, tick-caused pathology in sheep has been due largely, if not entirely, to tularemia. The evidence leading to this conclusion is presented in this paper. All the several outbreaks herein reported were personally investigated by Dade or by Dade and Dr. W. T. Huffman, the local representative of the federal Bureau of Animal Industry.

The results of the observations in Montana have been less definite and suggest that perhaps both tularemia and tick paralysis have been concerned in that state. The data relative thereto have recently been reported by Parker and Butler in the Seventh Biennial Report of the Montana State Board of Entomology.[5]

We are indebted to Surgeon Edward Francis, of the Hygienic Laboratory, at Washington, for the bacteriological tests, herein reported, which resulted in the recovery of *Bact. tularense* from the spleens of guinea pigs that had been injected with ticks and tissues secured from affected sheep, and which confirmed the diagnoses made at the Hamilton Field Station.

Idaho Observations Prior to 1928

The first known instance of tick-caused pathology in sheep in southern Idaho occurred in 1919, in a band belonging to the MacMillan Sheep Company, of Mountain Home. A loss of 250

lambs was experienced among sheep that were being grazed just north of the Indian Creek Reservoir, south of Boise.

The next occurrence was in the spring of 1920, when there was an extensive outbreak near Nampa.

In 1923, trouble was experienced in areas tributary to Shoshone and Mountain Home. In April of that year Dr. Herbert Dean, of Shoshone, gave a description, at the Boise meetings of the South Idaho District Medical Society, of an apparently tick-caused condition in sheep in his locality. Following these meetings, Parker, who had been detailed to attend them, went to Shoshone to investigate. It was found that the trouble was past. However, numerous bands had been affected. One owner, B. Darrah, had lost 150 ewes; another, J. C. Newman, 90 lambs. The aggregate loss had been considerable. The outbreak near Mountain Home was observed by Dade and Huffman. Sheep belonging to several owners were affected. Two of them, John Mandiola and a man named Riley, experienced heavy losses of lambs. The former lost 1300 lambs from ten bands, in some of which the loss amounted to ten per cent.

In 1924, there was a much less severe and less extensive recurrence in the Shoshone area. The locality was again visited, incidental to a more extended trip, by Surgeon R. R. Spencer and Parker from the Hamilton station. Again it was too late for worth while observations. There was opportunity, however, for a postmortem examination of two lambs from a band which had been affected. One was recently dead; the other was walking stiff-legged, was apparently ill and was killed. The only gross pathology found was the enlargement or slight injection of some of the regional lymph-glands. Glandular tissue and pieces of other tissues were placed in glycerin and forwarded to Hamilton where they were emulsified in salt solution and injected into guinea pigs. Unfortunately, it was necessary that these tissues be tested by an assistant who was not familiar with the lesions of tularemia, and although his autopsy notes for part of the guinea pigs injected with glandular tissue were suggestive of tularemia, the results lacked diagnostic value.

There were no reports of further outbreaks until 1927. In the spring of that year the most serious outbreak yet experienced occurred in country tributary to Arco, where nearly every band of a large number that totalled approximately 150,000 sheep was affected. The locality was visited by Dade and Dr. Huffman, May 9 to 11. The heaviest loss, in some instances amounting

to 5 per cent, was in the yearling bands. In those consisting of ewes and lambs, the lambs were the more severely affected, the loss among ewes being negligible. The total loss by death was estimated to have been between 3000 and 5000 head, principally yearlings. The economic loss among yearlings and ewes, aside from death, was considerable, the loss of weight often being 25 to 35 per cent. The ticks, which usually attach only to the less woolly portions of sheep, that is, at the base of the ears, on the sternum and in the axillae, were found generally distributed in the thick wool on the neck, shoulders, sides, the outer portions of thighs, and in much less numbers on the denuded parts. No autopsies were made. The inner side of recently removed pelts showed multiple hemorrhagic spots, corresponding to sites of tick attachment. These varied in color from light red to dark purple and in size from the diameter of a pea to that of a silver dollar. The temperatures of many sheep were found to range from 105.6 to 107.8°F. Scouring was frequent. Unfortunately no ticks or tissues were preserved for laboratory tests.

Trouble was experienced also in 1927 near Mountain Home, in several bands belonging to the Thompson Brothers. These bands were in the same general area in which the heavy losses occurred in 1919 and 1921 (see above) and in which our observations were made in 1928. A considerable number of lambs were lost but after removal of the sheep from the wood tick-infested sage brush to tick-free range, as advised by Dade, no more losses were incurred.

Idaho Observations in 1928

In 1928, a band belonging to H. Blackwell became seriously affected in the same area as the Thompson Brothers bands in 1927. The owner was unable to secure tick-free range and a heavy loss resulted. Also the Thompson Brothers bands were again affected; but at the first signs of illness they were removed to higher ranges and, as before, further loss was thus prevented. These bands were visited by Dade and Dr. Huffman, and later by Dade and Parker. It was from them, especially the Blackwell band, that evidence was secured that points to tularemia as at least the usual cause of trouble in southern Idaho. A detailed account of the investigation follows:

Blackwell band: This band consisted of 900 old ewes, 300 yearlings and 700 lambs. They had wintered at Grandview south of Mountain Home and, in the early spring, were purchased

by H. Blackwell. Delivery was made at Mountain Home about the middle of March. Thence they were trailed by way of Orchard and Round Butte to Black Creek, fifteen miles south of Boise, where spring range had been leased. While en route they traversed a section near Round Butte and Orchard in which the losses had been experienced by the Thompson Brothers bands (see above) in 1927, and where bands of the same owners were again affected in 1928. The loss of lambs was noted for several days prior to reaching Black Creek and immediately on arrival they called on Dade for assistance and the band was visited on April 1, by Dade and Dr. Huffman. Dade recommended the removal of the band from the sage brush to a tick-free area, but local range of this character all being occupied, the owner was forced to hold his sheep on Black Creek. It was therefore necessary to treat each affected animal individually, the ticks being either hand-picked or Kreso dip applied to points of attachment. Affected animals with temperatures of from 106.0 to 107.8°F. were numerous. Many were scouring. The band was again visited by Dade and Parker, April 12, and though much improved, losses were still being experienced and the band showed marked loss of condition. Three ewes were "down," several recently dead lambs were seen, and there were about twenty sheep that showed by high carriage of the head, by stiff-legged walk or the presence of scours, that they were affected. Up to this date, fifty per cent of the lambs, twenty-five per cent of the yearlings, and a smaller percentage of ewes had been affected. Sixty lambs and fifteen ewes had died.

Dead jack rabbits were reported as numerous along the trail followed to Black Creek and on the Black Creek range.

Materials secured for test: On April 1, Dade and Dr. Huffman secured both ticks and glandular tissues from two lambs (one of them recently dead) and, on April 12, Dade and Parker secured tissues from a lamb recently dead, ticks from an old ewe acutely ill, ticks and blood from a yearling and from an old ewe acutely ill, and blood from a lamb and old ewe both of which had recovered. The tissue samples were placed in glycerin and with the ticks were forwarded to the Hamilton Laboratory, where the necessary tests for infectiousness were made.

1. Lamb: April 1. Acutely ill; down and unable to rise. Killed and autopsied. Ticks recovered.

Tissue: April 4. A salt solution emulsion of pooled prescapular, cervical and postpharyngeal glands was injected subcutaneously into two guinea pigs. Both were dead April 9, with typical lesions of tularemia.

Ticks: April 4. Two engorged females were injected subcutaneously into each of two guinea pigs, and three partly fed females were placed on a single guinea pig for feeding. Both tick-inoculated pigs died typically April 8. On April 17, the guinea pig on which the ticks were fed was still alive but very sick and was killed and autopsied. Only one tick had fed. There were typical inguinal buboes and spleen was enlarged five times. Infection was apparently subacute and transfer would certainly have produced typical lesions.

Bacteriological confirmation: On April 8, the spleens of the two guinea pigs that had been injected with engorged female ticks were forwarded to Dr. Francis, and from them both he recovered *Bact. tularense* in pure culture.

2. Lamb: April 1. Dead ten or twelve hours. Autopsied. Ticks recovered.

Tissue: April 4. Pooled tissues, tested as for lamb 1, caused the death of two guinea pigs of typical tularemia, respectively, April 7 and 8. From one, spleen emulsion transfers were made to four other guinea pigs, all of which died typically in two to four days.

Ticks: April 4. Two engorged females were each injected subcutaneously into a gunea pig. Both died of typical tularemia, April 9. Two partially-fed ticks were placed on a third guinea pig which also died typically twelve days after infestation.

Bacteriological confirmation: On April 8, the spleen of one of the guinea pigs injected with pooled gland emulsion from lamb 2 was forwarded to the Hygienic Laboratory at Washington. A pure culture of *Bact. tularense* was recovered from inoculated guinea pigs. A strain started from this culture is still being maintained at the Hamilton Station.

3. Yearling: April 12. According to herder, this yearling had been walking lame for several days. Blood sample and ticks secured.

Ticks: April 16. Three guinea pigs were each injected subcutaneously with two partially to fully engorged females. All were dead of typical tularemia on the sixth day, April 22.

Agglutination test: May 4. Complete agglutination of *Bact. tularense* occurred in a dilution of 1:1280 and was partial at 1:2560.

4. Lamb: April 12. Recovering, but still showing evidence of illness by loss of weight and carriage of head. Blood sample only.

Agglutination test: May 4. Complete for *Bact. tularense* in all dilutions up to and including 1:640, partial at 1:1280.

5. Old ewe: April 12. Recovered, but was coughing and showed loss of weight. Blood sample only.

Agglutination test: May 5. Complete for *Bact. tularense* in all dilutions up to and including 1:160.

6. Dead lamb: April 12. Dead not over one day. Autopsied by Dade and Parker and specimens taken of prescapular, cervical and postpharyngeal glands and of liver and spleen. The prescapulars and postpharyngeals were injected, though not markedly, and the former were one-half enlarged. Ticks had detached and crawled away but there was tick excrement in the wool and the hide showed numerous points of attachment.

Tissue: April 16. Tested by inoculating subcutaneously into guinea pigs.

Prescapular glands: Three guinea pigs were injected. All were dead of typical tularemia April 23, seven days after inoculation.

Postpharyngeal glands: Three guinea pigs injected. April 23, all were dead of typical tularemia.

Cervical glands, spleen and liver: Three guinea pigs were injected with each tissue. All were negative, as were also transfer guinea pigs inoculated with spleen and liver emulsions from the initial test animals.

7. *Old ewe:* April 12. One of the three noted as being down. Had apparently been down several days. Ticks had been soaked with Kreso dip; many were still attached under the jaw, around base of ears and on side and back of head. They were misshapen, were apparently unhealthy, were feeding but little, and many were either dead or nearly dead. Most of female ticks were but slightly engorged, none fully. Scouring had been severe. Unlikely to live. Ticks and blood sample secured.

Ticks: April 16. Four guinea pigs, each injected with two slightly-fed females, remained afebrile and were apparently negative when killed and autopsied, May 4, eighteen days after inoculation.

Agglutination test: April 30. Complete for *Bact. tularense* in a dilution of 1:1280.

April 12. Same data as for old ewe 7, except that condition not quite so bad. Ticks were less numerous but with same unhealthy appearance. Blood sample only.

Agglutination test: May 5. Complete for *Bact. tularense* in all dilutions up to and including 1:640, partial at 1:1280.

Summary of Blackwell band data: Table I summarizes the data for the Blackwell band. Plus and minus signs indicate tests which were respectively positive and negative for tularemia; numerals show the number of tests of each material, and blanks that no material was available for the test indicated.

The table shows that positive tests indicative of present or recent infection with *Bacterium tularense* were secured from each of the eight sheep concerned and that these findings were confirmed bacteriologically in three instances.

Dead lambs (2 and 6): Tissue was tested from two dead lambs, both dead less than 24 hours. Glandular tissue from both was infectious, the test material from one being a pooled emulsion of prescapular, postpharyngeal and cervical glands, while from the other these glands as well as a piece of the spleen and liver were tested separately. The pooled glandular tissue of the former was positive. For the latter the postpharyngeal and prescapular glands were infectious, but the tests of the cervical glands and of the spleen and liver were negative. Three groups of ticks from the first also infected the test guinea pigs, two by subcutaneous

TABLE I—*Summary of data on Blackwell band*

SHEEP TESTED		TISSUES					TICKS	AGGLUTINATION OF BACT. TULARENSE
		GLANDS			LIVER	SPEEN		
		PRE-SCAPULAR	POST-PHARYNGEAL	CERVICAL				
Dead	2. Lamb, dead 10 or 12 hours	Glands pooled 2+*					3+ 2 by inoculation 1 by feeding	
	6. Lamb, dead less than 24 hours	3+	3+	3—	3—	—		
Acutely Ill	1. Lamb "down," killed, autopsied	Glands pooled 2+					3+* 2 by inoculation 1 by feeding	+ 1:2560
	3. Yearling not "down"						3+ by inoculation	+ 1:1280
	7. Ewe, old, "down," dying						3— by inoculation	+ 1:1280
	8. Ewe, old, "down," might recover							+ 1:1280
Recovered	4. Lamb, just recovering							+ 1:160
	5. Ewe, old, recovered							

*Culture of *Bact. tularense* recovered from test guinea pigs.

inoculation, one by feeding. From the latter no ticks were secured.

Pure cultures of *Bact. tularense* were recovered from the spleens of two guinea pigs injected with ticks from lamb 2.

Sheep acutely ill (1, 3, 7 and 8): Materials from four acutely ill sheep were tested, a lamb (1), a yearling (3), and two old ewes (7 and 8). Tissue was tested from only one of these sheep, i. e., the lamb (1) which was down and was killed and autopsied on April 1. Two guinea pigs inoculated subcutaneously with pooled glandular tissue and three others, two inoculated with two partially-fed females each, and one on which three ticks were fed, all died of tularemia. From the three other sheep (3, 7 and 8) samples of serum were secured, all of which agglutinated *Bact. tularense*, respectively up to and including dilutions of 1:2560, 1:1280, and 1:1280. From both the yearling and one of the old ewes (which was down), three groups of ticks of two partially-fed females each were inoculated subcutaneously into guinea pigs. Those from the yearling infected all three guinea pigs, while those from the ewe (see below) caused no lesions suggestive of tularemia.

A pure culture of *Bact. tularense* was recovered from the spleen of one of the guinea pigs inoculated with glandular tissue from lamb 1.

Recovered sheep (4 and 5): The sera from blood samples from two recovered animals, a lamb shortly recovered (4) and an old ewe longer recovered (5), agglutinated *Bact. tularense* respectively in all dilutions up to and including 1:640 and 1:160.

The above tests of eight affected animals show (a) that tularemia-infected tissues and ticks were recovered both from dead, and from acutely ill sheep, (b) that sera of both acutely ill and of recently recovered sheep agglutinated tularemia antigen in high dilution, and (c) that *Bact. tularense* was recovered in pure culture from sheep tissues and from infesting ticks. The negative test of the ticks from ewe 7 may have been due to one of the following possible causes: (1) the ticks responsible for the condition of the ewe may have dropped off or have been killed by the Kreso dip, (2) the responsible ticks may not have been recovered since only a few ticks were taken for test, or (3) the infectiousness of the bacterium, if originally present in the ticks, may have been destroyed by the dip (this has been known to happen to Rocky Mountain spotted fever virus in infected ticks soaked in chloroform) which had plainly affected the vitality of the ticks.

Thompson Brothers bands: Three bands belonging to the Thompson Brothers of Mountain Home were started to graze in late March in the same section, near Orchard and Round Butte, through which the Blackwell band passed. Animals of all three bands, especially the lambs, soon showed the symptoms characteristic of trouble from ticks, but the owners having had experience in the same area in 1927 and having had the benefit of Dade's advise as to what to do, immediately moved their sheep out of the sage brush to a tick-free range. The extent of loss was not known other than that it was limited to a small number of lambs. It was possible to reach only one of these bands which was visited by Dade and Parker on April 14. By that time most of the animals which had been affected had so far recovered that they could not be distinguished from the remainder of the band. However, blood samples were secured from two lambs, a black one that the herder definitely knew had been affected, the other a lamb that stumbled when walking and which when caught was found to have a considerable area of necrotic tissue in the left axillary region, and, in the right, a mass of indurated tissue surrounding a healing abscess. Both sera agglutinated *Bact. tularense* in dilutions up to and including 1:640.

Symptoms and Gross Pathology

Dade and Huffman have observed the following symptoms in the several outbreaks in southern Idaho reported above. Onset is usually slow and the first suggestive symptoms are a stiff-legged walk and high carriage of the head, both presumably due to irritation caused by involvement of regional glands, and stupor. Affected animals will be seen in groups on the edge of the band and when trailing are frequently on the tag end. No incoöordination of leg muscles has been observed as reported in tick paralysis. There is usually, but not perhaps always, an elevation of temperature, which may reach 107.8°F. The pulse beat is rapid and sometimes almost imperceptible. Respiration may be accelerated, especially in animals with high fever. Scouring is frequent, and probably always occurs in fatal cases. Urination is frequent and scanty. Reflexes are normal. Pupils of eyes may be somewhat dilated. Toxemia and depression are marked. A cough often develops which persists during recovery. Severely affected animals soon become so weak that they are unable to stand. Death may occur within a few hours after going down, or not for several days. Sheep that are down, even advanced cases,

when turned on their side will invariably struggle and throw their legs about in an effort to right themselves. If infesting ticks are removed or treated with a tick-killing agent soon after animals are down, recovery is more likely. Removal after animals are down some time is of no value.

The infesting ticks are most often attached about the base of the ears, on the top of the neck and under the fore legs, but sometimes are more widely distributed over the body even attaching in the heavily wooled portions.

The flesh side of hides removed from animals that have died almost invariably show reddish to purplish hemorrhagic areas at the sites of attachment of some, but not of all, the infesting ticks. These areas vary in size from that of a pea to that of a silver dollar or even larger. Hadwen[1] has noted the same condition in British Columbia in sheep affected with tick paralysis, so the condition is not specific, nor for that matter is it known to what extent such areas may mark the site of attachment of ticks on sheep not pathologically affected. It is certain, however, that only a relatively small proportion of infesting ticks produced these areas.

On some affected sheep there is an induration of tissue or local ulceration and necrosis at the sites of attachment of some of the ticks. This is particularly true of the axillary regions, where the tissue involvement is sometimes quite extensive.

Dade and Dr. Huffman have autopsied numerous affected animals on the range. The only gross pathology observed has been the enlargement or injection of regional glands of the anterior portion of the body; that is, the portion where ticks are most frequently attached.

Discussion

The initiating role played by the wood tick in the above described pathology in southern Idaho sheep is indicated by the following data:

1. That it occurs only during the season that wood ticks are active, and appears in the early spring soon after the sheep are placed on the tick-infested sage brush range.
2. That in an affected band a high percentage of infection was present in wood ticks recovered from affected sheep and that epidemiological evidence points to the wood tick as the only likely transmitting agent concerned.

3. That when affected bands are moved from tick-infested areas to tick-free range there is an abrupt drop in the incidence of new cases, which soon cease entirely.
4. That when affected sheep become so ill that they cannot stand, they usually recover if the infesting ticks are removed or treated with some tick-killing agent soon after the animal is down; while if the ticks are not molested recovery is far less frequent.

We believe that the disease condition concerned is due to *Bact. tularense* for two reasons; first, because of the data secured from the Blackwell and Thompson bands (especially the former) in the spring of 1928, and second because the symptoms, as observed by Dade and Dr. Huffman in affected bands since 1919, have consistently been the same as those noted in the Blackwell band, in which each of eight acutely ill, recently dead, or recently recovered animals gave evidence of present or recent tularemia infection.

The Blackwell band data as summarized in this paper show that: (a) tularemia-infected tissues and ticks were recovered from both dead and acutely ill sheep, (b) that sera of both acutely ill and recently recovered sheep agglutinated tularemia antigen in high dilution (not less than 1:160 in the case of recently recovered sheep, and as high as 1:2560 in animals acutely ill), and (c) that *Bact. tularense* was recovered in pure culture from tissues from an affected lamb and from infesting ticks. Of the Thompson Brothers band, which became affected in the same area as the Blackwell band, but was not seen until after removal to tick-free range, the sera of two lambs—one fully recovered, the other still showing evidence of recent illness—both agglutinated *Bact. tularense* in a dilution of 1:640.

It seems extremely unlikely that eight acutely ill, recently dead, and recently recovered sheep, selected more or less at random from the Blackwell band, which is known to have been heavily infested with an infected tularemia-transmitting parasite, could all have given the recorded evidence of recent or present infection with *Bact. tularense* and this organism not have been the immediate cause of the pathology noted.

Unfortunately no blood samples of acutely ill sheep were tested for infectiousness. However, the fact that of four chance-selected ticks tested from lamb 2, a minimum of three (75 per cent) were shown infected; that of five similarly selected ticks

from lamb 1 (seven were used, but two did not feed) a minimum of three (60 per cent) were shown infected, and that of six such ticks from yearling 3, a minimum of three (50 per cent) were shown infected, suggests that a part at least of these chance-selected ticks may have derived their infection from the blood of their respective host sheep. The apparent alternative would be that all had acquired their infection from rodent hosts on which these ticks engorged as nymphs during the spring and summer of 1927. In order that even 50 per cent have been infected—since nymphs are continually engorging from early spring to early fall—it would apparently have been necessary that a very high percentage of their rodent hosts (principally jack rabbits in the area concerned) have had a chronic bacteremia. This may not be an impossible condition, but it is certainly an unlikely one.

Furthermore, the probability that the percentage infected was actually higher than the minimum indicated is shown by the fact that six of the nine positive tests concerned were of two ticks each and three of one tick each. That each of the six pairs contained but one infected tick is extremely improbable. In this connection attention is called to the Montana data of Parker and Butler[6] who found a minimum of 40 per cent of ten female ticks, taken from two ewes suspected of being ill with tularemia, to be infected. These were tested in pairs, four tests being positive for tularemia, while the fifth was terminated by intercurrent infection. That a reasonably high percentage of *unfed* adult ticks may be found infected has been indicated by the result of tests of such ticks collected near Bozeman in the spring of 1925,[6] but no known data have indicated such high percentages as those here concerned unless the ticks were taken from a tularemia-infected host.

Although it seems certain that *Bact. tularense* is responsible for the above-described pathology in southern Idaho sheep, there are attendant conditions for which fully substantiated explanations are not apparent:

(1) Why is it that, as shown in the records above, sheep of all ages are not equally severely affected? For example, the losses in the Mandiola and Newman bands in 1923 were, as far as our data show, limited to lambs, the ewes in the same bands evidently having been but slightly affected or not at all. That same year, in the Darrah bands, grazing in the same general country as those of Newman, only yearling ewes were lost. In the severe outbreak near Arco, in 1927, the heavy loss was among yearlings, the lambs

being affected but little and the ewes even less. In the Blackwell band in 1928, the heaviest loss was among the lambs although the yearlings and ewes were also affected—the ewes more than the yearlings. It is a natural first thought to imagine that this apparent difference in the relative susceptibility of lambs, yearlings and ewes, exposed to the same source of infection, might be explainable on the basis of relative degrees of acquired immunity. This, however, seems to necessitate the assumption that during the years between outbreaks sheep are exposed to a less virulent type of tick-borne tularemia, capable of inducing mild but immunizing reactions. While this may not seem an entirely unreasonable hypothesis, the evidence at hand does not support it. Moreover, if relative degrees of immunity were concerned, it would seem probable that whenever an outbreak occurred the lambs, which would have had no opportunity to acquire immunity, should always be at least as severely affected as the older sheep; yet, the evidence of the Darrah bands, in 1923, and of the Arco outbreak, in 1927, shows that this is not the case. Nor can it be suggested to explain this point that lambs have a higher degree of natural resistance than older sheep, since in some of the instances cited they have been the ones most severely affected.

This discussion invites consideration of the susceptibility of sheep as shown by laboratory experiments. The first attempt to infect them was made by McCoy and Chapin,[7] in 1911. Five sheep were tested (no age data given), four by the subcutaneous injection of salt solution emulsions of spleen, liver and other tissues from recently dead guinea pigs, and one by vaccination with an infected spleen. Three of the injected sheep were infected and two died; while one that received only a small amount of emulsion and the one vaccinated, both remained well. Parker, in the course of studies at Hamilton, has three times attempted to infect yearling sheep (in prime condition), one by the feeding of infected ticks, and two by the repeated daily rubbing of infectious tick excrement into the denuded axillary region (where further rubbing of the contaminated area was assured by movements of the leg). The yearling on which ticks were fed remained well, and serum samples taken 14 and 21 days after the start of tick feeding, both failed to agglutinate *Bact. tularense* even in low dilution.[6] Of two sheep that were rubbed with tick excreta emulsion, the serum of one agglutinated *Bact. tularense* in dilution of 1:80 nineteen days after the first application and in

dilution of only 1:10 both 123 and 217 days after the last application. The serum of the second caused agglutination of *Bact. tularense* in dilution of 1:160 forty-seven days after the first application and in dilution of 1:40 twelve days after the last application. Except that the second of the two sheep rubbed with tick excrement showed some loss of weight and was less lively than its mate none of these three animals showed apparent evidence of illness. The sera of these sheep taken before test all failed to agglutinate *Bact. tularense*. Francis[8] records the death in 23 days of a sheep injected with a pure culture of *Bact. tularense*. In correspondence with Parker, Dr. Francis has further advised (a) that the serum of this ewe (4) agglutinated *Bact. tularense* in a dilution of 1:10 before inoculation and in dilutions of 1:1280, 1:2560, 1:2560, and 1:640 respectively on the tenth, fifteenth, seventeenth and twenty-third days after inoculation; (b) that abortion occurred on the fifth day; (c) that its temperature was 108.0°F. on the day after inoculation and was 105.8°F. or over until the sixth day, after which decline was gradual until death; (d) that skin at site of inoculation was slightly thickened but without inflammation, that spleen and liver were apparently normal and lungs appeared normal except for several areas of red consolidation about one inch in diameter, and that sections of spleen, liver and lungs showed no microscopic pathology. A second sheep (2) that was similarly inoculated had a temperature of 107.6°F. the day following, and its temperature was 105.8°F. or above on all but one day until the tenth day; its serum agglutinated *Bact. tularense* in dilutions of 1:320 and 1:80 respectively on the fifteenth and twenty-first days, the last sample having been taken just before death; at autopsy, spleen, liver, lungs and glands appeared normal.

These data are too few to be of great significance, but they do show that fatal infections have occurred in laboratory-infected sheep, while on the other hand, they suggest that some sheep, at least, are more or less naturally resistant.

(2) The second puzzling point is that the outbreaks of tick-caused trouble, as thus far observed, have occurred only during the early part of the tick season, very shortly after sheep have been driven on to the range and early in the wood-tick season. The latest outbreak was that near Arco in the early May of 1927. This lateness is more apparent than actual, since the sheep concerned, which had been grazing on tularemia-free range earlier in the season, did not reach the high country around Arco till

shortly after the local ticks had appeared. It is possible that this apparent restricted seasonal occurrence may result from the fact that, due to lack of water, the great bulk of sheep are moved early in season from the sage brush desert areas to ranges at higher altitudes where wood ticks are usually far less numerous or altogether absent. In spite of the reasonableness of this explanation, it is not yet clear that it is to be accepted as more than a contributing factor.

Another possibility is that the virulence of *Bact. tularense*, as it occurs in the wood tick, decreases as the tick season advances and, except early in the spring, is not sufficiently great to overcome the apparent natural resistance of sheep. In this connection, laboratory observations made at Hamilton have shown that tularemia infection is far less persistent in wood ticks than the virus of Rocky Mountain spotted fever. The likelihood that early shearing, as practiced in southern Idaho, might be concerned has also been considered—ticks are much less troublesome on sheep after shearing—but this seems to be excluded by the fact that although lambs are not sheared, there is no more evidence of late trouble among them than among older sheep.

(3) A third point which is not clear is why the removal of the wood ticks from an affected sheep (or killing them *in situ*) *after* the infectious process has been initiated is an aid to the recovery of the host. The only explanation we have to offer is that normal sheep have a certain degree of natural resistance which is broken down only because of the protracted inpouring of infection that results from the prolonged feeding of infected female ticks and that when this stream of infectious organisms is shut off by tick removal, before resistance has been completely overcome, then recovery follows. In this connection it would be of interest to determine whether, as in tick paralysis, the removal of females only, would be as effective as the removal of all ticks. This has been true of tick-affected sheep in eastern Montana, but it was uncertain whether the condition concerned were tick paralysis or tularemia.[6]

Sufficient tests of the tissues of infected sheep have not been made to indicate the usual distribution of the infectious agent. In the tests of McCoy and Chapin, infection was recovered from the spleen of one of their two sheep that died, but no other organs were tested. From the other, guinea pigs were injected with material from the site of inoculation and from a bubo, and

with spleen tissue and heart-blood. Only the one injected with material from the site of inoculation became infected.

In our own tests with sheep of the Blackwell band, an injection of an emulsion of pooled prescapular, cervical and postpharyngeal glands of each of two lambs proved infectious, while of a third, prescapular and postpharyngeal gland emulsions proved positive, but those of the cervical gland and of spleen and liver were negative. No other tissues were tested.

Of the two sheep infected by Francis, infection was recovered from the blood of one on the sixth day after inoculation but not from samples taken on the tenth and fifteenth days; the blood of the second was negative on the fifteenth, sixteenth, seventeenth and twenty-first days.

These observations recall the results of Parker and Francis[9] in 1925, with tissues of coyotes dead of tularemia.

From one of these coyotes infection was recovered from the salivary and axillary glands, and the spleen; from another, only from the prescapular glands, and from a third, only from the salivary glands. Of all three coyotes the salivary, axillary and inguinal glands and the spleen were tested and in addition, the liver of two, the lungs from one, the prescapular and retroscapular glands from one, and heart-blood from one. All the tests were apparently negative except as noted.

The striking feature of these observations has been the death of both sheep and coyotes from apparent tularemia infection, but without marked gross pathology such as is so characteristic of the disease in laboratory animals. In part of these animals, there seemed to have been only a local invasion of tissue by the bacterium, but the evidence of extent of invasion is admittedly scanty.

A comparison of the outbreaks of wood-tick-caused pathological conditions in eastern Montana sheep (Parker and Butler[6]) with those in southern Idaho sheep would be of interest, but unfortunately, the very incomplete nature of the Montana data precludes the mention of more than a few points. Seasonal occurrence is the same. In Montana, affected sheep recovered if only female ticks were removed. In Idaho, this has not been tried, but the removal or treatment of all ticks was found to be of value. In Idaho affected animals frequently ran high temperatures, while in Montana there is no evidence as to whether or not rises in temperature occurred. In the latter state also there are no data as to the occurrence of scouring. In the Montana band

(Danford) concerning which there was the most reason to believe *Bact. tularense* to have been the disease-causing agent, sick ewes aborted their lambs.[6] This occurred in other tick-affected bands as well. It has not, however, been reported in Idaho where lambing for the most part takes place early in the season before the sheep become tick infested. Late lambing ewes are taken to the high ranges for lambing and thus escape tick infestation. Abortion in an experimentally infected sheep was observed by Francis as reported above.

There is no evidence that tick paralysis, as it is described by Hadwen[1] and Bruce,[2,3] has played any part in the southern Idaho trouble. There is, however, no apparent reason why it should not occur there, especially if, as is generally held, this condition is caused by a toxin secreted by the salivary glands of engorging female ticks.

The proven occurrence of *Bact. tularense* in the tissues of sheep opens the question of the possibility of human infection from the handling of infected carcasses. Infection is definitely possible through the primary contamination of the hands with the tissues of crushed infected ticks held in the wool or with tick excrement which is commonly present in large masses. The fingers might also become contaminated by contact with necrotic tissue which sometimes develops at the points where infected ticks have been attached. The chance that infected meat might reach the markets and be a source of danger to persons in slaughter-houses, packing-houses and to the consuming public seems less likely, but cannot be altogether dismissed, especially if animals are slaughtered for immediate local consumption.

Further and more carefully planned field and laboratory observations of wood-tick-caused tularemia in sheep are desirable: (1) to determine the extent to which it is concerned in wood-tick-caused pathological conditions, (2) to determine the geographical limits of the occurrence, which certainly are wider than indicated by present data, (3) to secure more detailed epidemiological, symptomatological and pathological data, and (4) to determine if the meat of infected slaughtered sheep is a possible source of human infection.

As a part of these investigations, affected bands and local resident tick populations should be continuously kept under observation throughout, at least, the duration of acute trouble (observations made thus far have been largely of an incidental nature), and the field data thus secured should be correlated with

the results of laboratory tests of sheep tissues, ticks and serum samples. Laboratory attempts to reproduce in sheep the condition concerned by means of experimentally infected ticks, or by ticks collected in an area where an outbreak is occurring, should also yield data of value.

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Bridge on Belle Isle, Detroit

MALIGNANT GROWTHS IN DOMESTIC ANIMALS*

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To most veterinarians a tumor is something to be treated; its type need not necessarily be diagnosed. This point of view, perhaps, is better appreciated if the relative economic importance of neoplastic diseases is compared to diseases of an infectious nature, such as contagious abortion, hog cholera and tuberculosis. Infectious diseases are quite different in their general pathologic manifestations from neoplasms in that many of them spread rapidly and affect large numbers of individuals, requiring constant vigilance on the part of veterinarians to keep them within control. Although the practitioner may be called on to treat many animals suffering from infectious diseases during a week, it is unusual indeed if he sees more than a few true tumors in the course of a year. Consequently, from the point of view of the veterinarian engaged primarily in the control and treatment of diseases of animals, the subject of neoplasia naturally occupies a place of minor importance. This attitude also obtains in most veterinary colleges in which little more than an introduction is given to the study of tumors. This is, of course, justifiable from the standpoint of time, since in the present badly overcrowded curriculum many other subjects of much more economic significance to the graduate than tumors are denied sufficient time for proper consideration.

Although the veterinarian, like the average physician, may not be fully informed concerning the pathologic and academic aspects of neoplasia, he is, I believe, none the less interested in its many biologic aspects. There is complete agreement that cancer in man and cancer in animals represent identical expressions of perverted activity of cells, and although there are minor differences of a clinical and morphologic nature, the disease in the respective species is quite comparable. Surprising as it may seem, many laymen do not know that animals can and do develop cancer capable of the same menacing possibilities as cancer in human beings. On the other hand, when the owner of an animal

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afflicted with cancer is aware of the close similarity between cancers of animals and those of man he often manifests a keener interest in the condition than he does in other diseases of far greater monetary importance.

The progress made in cancer research has been due in a large measure to studies pertaining to tumors of animals, many of which possess a peculiar and, perhaps, remarkable quality for transmission from one animal to another, usually of the same species. The extreme importance of cancer in animals, in the final solution of the cancer problem, is impossible to estimate and as veterinarians we can do much in the accumulation of valuable information that will materially further the knowledge pertaining to this little understood phenomenon.

Incidence

In a discussion of this kind it is usually proper to say something concerning the incidence of tumors as they occur in the lower animals. Unfortunately statistical data are too meager to enable one to obtain a comprehensive understanding of this phase of the problem. As veterinarians we are far behind our sister profession of medicine in this regard, due, of course, to the great interest manifested by physicians and laymen alike in malignant disease. A conservative estimate puts the annual number of deaths of human beings in the United States, as a result of cancer, at somewhat more than 100,000. This fact, together with the system of registration of deaths, and the increase in the number of necropsies, makes it possible to determine with some degree of accuracy the incidence of tumors in human beings.

Owing to the lack of a central agency where causes of spontaneous deaths of animals can be analyzed, we are denied much of statistical value as regards tumors. Further, most of the meat-producing animals are slaughtered before the age at which malignant tumors are most likely to develop, making it impossible to estimate the incidence of neoplastic disease if these animal were permitted to attain a longer life.

That the animals killed for meat yield only relatively few neoplasms is shown by the figures compiled from the report of the Chief of the Bureau of Animal Industry of the United States Department of Agriculture for 1926 (table I). In the report of the inspection of carcasses in abattoirs supervised by federal meat inspectors an attempt is not made to differentiate condemnations made for true tumors and for abscesses. In other words,

all swellings, whether neoplastic or inflammatory in nature, are listed as tumors. Consequently, the percentage of neoplasms encountered in packing-house material is actually lower than the figures in the tabulation would indicate.

TABLE I—*Condemnations for tumors and abscesses*

ANIMAL	CARCASSES PASSED	CONDEMNED FOR TUMORS AND ABSCESSES	
		CARCASSES	PARTS
Cattle	9,994,485	1,797	3,940
Calves	5,299,841	52	431
Sheep	12,339,763	168	35
Swine	40,299,704	2,438	238,788

The paucity of neoplasms in young individuals is suggested in comparing the figures for calves with those of adult cattle. Sheep, as a class, are also relatively insusceptible to neoplastic disease, and the data in table I substantiate the observations of many others in this regard.

The relatively large number of condemnations of carcasses of swine, and parts of carcasses, cannot be attributed to a high incidence of tumors, but must be explained on the basis of abscess formation to which the hog is particularly susceptible following the wounds of vaccination and castration. Even the most common tumor in swine, embryonal nephroma (Wilm's tumor), occurs only in about one case in 40,000.

The use of horse flesh for food has made it possible to obtain reliable data concerning the incidence of cancer in this animal. Martel,[9] Chief of the Sanitary Veterinary Service of Paris, studied this problem from material secured from 39,800 horses slaughtered in the abattoirs of that city. In this series, 184 animals were affected with carcinoma; the majority of them were more than fifteen years of age. From these data it is evident that malignant epithelial tumors were present in about 0.5 per cent of the animals and since cancer alone is mentioned the total incidence of tumor must have been higher. In my own series of thirty-one neoplasms of all varieties in horses, sixteen (approximately 50 per cent) were carcinomas. Of the 184 carcinomas in Martel's series, sixty-two were in kidneys, fifty were in testicles, and forty-five were in mammae. Carcinomas affecting the eye or penis were not mentioned; although these organs were most frequently involved in the cases which came under my observation.

Fröhner,[5] at the Berlin Veterinary High School, found tumors in 69 per cent of 5000 horses, and 13 per cent of them were carcinomas. He also found, in a series of more than 60,000 dogs, that almost 3000 had some form of tumor, more than 1100 being true carcinoma. Cohrs,[1] in a total of 737 necropsies on dogs, found tumors in seventy; Goodpasture,[6] and Goodpasture and Wislocki,[7] recorded the finding of tumors in every one of fifty dogs examined. These, however, were old animals and it has been the experience of many observers that neoplasms occur in a surprisingly large number of dogs of advanced age. The frequency of the occurrence of malignant tumors in cattle is suggested by Trotter's[11] figures. He found 131 malignant neoplasms in an examination of 47,362 cattle slaughtered during one year (1903) at Glasgow.

The one animal for which it should be possible to assemble more or less reliable data on the occurrence of tumors is the common fowl. The average normal death-rate among fowls is relatively high and necropsy is performed easily and rapidly. The large number of fowls constituting many of the present-day flocks offers an extremely worth while opportunity for the pathologist to study avian neoplasia in all of its various phases. The poultry industry has attained such an important position commercially that the owner of poultry is keenly interested in every condition which may affect the well-being of his flock. Consequently, large numbers of fowls come to necropsy and a great many internal neoplasms are encountered.

Curtis[2] reported on the frequency of the occurrence of tumor in the domestic fowl which had been noted in necropsy data over a period of eight years at the Maine Agricultural Experiment Station. Of the 880 birds examined, tumors occurred in seventy-nine (8.96 per cent). The apparent influence of age on the incidence of tumor was noticeable. In birds less than two and one-fourth years of age, the incidence of tumor was 7.37 per cent, while in older birds the incidence was more than 19 per cent. Most of the fowls on which necropsy was done were hens, and in a large number of them the ovary and oviduct were affected with neoplasms. Unfortunately histologic studies were not made of Curtis' material, and hence it loses some of its value to the pathologist.

Schneider (1926)[10] reported on the incidence of tumor in about 11,000 pullets between the ages of six and eighteen months, all of the same age group and subject to the same general conditions

of management. Many breeds were represented. Data were obtained over a period of four years and the death-rate from tumor was found to be between 2 and 3 per cent. Since only the birds that died spontaneously were examined, the total incidence of tumor would be considerably higher if it were possible to include the birds affected with neoplasms which did not cause symptoms or death.

Ehrenreich[3,4] found seven malignant tumors among 4000 chickens intended for food; five were carcinomas. Again the influence of age on the occurrence of neoplasms in chickens is suggested. All of the growths occurred in old hens, and in 3000 of the birds examined which were less than one year of age, tumors were not found.

I think almost every poultry pathologist will agree that fowls, in surprisingly large numbers, are victims of tumors, and although reliable statistics dealing with this phase of incidence are scarce, the percentage must be high, higher in fact than in mammals.

In the examination of 1800 guinea pigs and 738 rabbits that died during the last year at the Institute of Experimental Medicine of The Mayo Foundation, only one neoplasm was found in guinea pigs, and none was found in rabbits. Apparently neoplasia is unusual in these animals, although such a conclusion can be only general.

The foregoing data, although not in any sense exhaustive, should be sufficient to emphasize the fact that tumors of the domestic animals are not uncommon but instead are rather common in most of the species. They are more common in animals that are permitted to attain maturity or old age, and I believe that if the lower animals were permitted to live out their natural span of life most of them would probably show a tumor incidence comparable to that of man.

Treatment

In the treatment of malignant growths the veterinarian has not kept pace with the physician. Too often the condition is considered hopeless after a cursory examination, and a valuable work animal or dairy animal is either permitted to live until it dies as a result of the disease, or it is slaughtered in the hope that something may be salvaged. Again, the victim may be a valuable and much esteemed pet which may be condemned to an early death without the benefit of the treatment that would probably be

accorded a human being suffering from a similar tumor. While I am not assuming that animals shall be given the elaborate and expensive therapeutic assistance often brought to the assistance of human beings suffering from cancer, I do make a plea for a better understanding of the general pathologic considerations of the tumors of animals to the end that the animals affected with malignant growths may receive the benefit of the information available, even if palliation only is afforded. Not all of the so-called malignant tumors are necessarily fatal, and the proper treatment of them may not only prolong the life and usefulness of a valuable animal, but apparent cures may be obtained in selected cases by appropriate means.

The rational treatment of neoplasms, like the rational treatment of any disease, depends largely on the making of a correct diagnosis. Simply to designate a given condition as a tumor is as unsatisfactory as referring to all acute abdominal disorders which result in pain as colic. Unfortunately, the diagnosis of internal neoplasms in animals has not achieved the refinement of technic practiced in the diagnosis of neoplasms of human beings, and, as a consequence, the majority of tumors, benign and malignant, which involve the internal structures of animals are usually not discovered until after death. Even if they were suspected, their inoperability in the larger species is apparent, and although operation may occasionally be resorted to for the relief of an internal tumor in smaller animals, the surgeon is much handicapped by his failure to receive the cooperative assistance from the animal on which success or failure so often depends.

As a matter of fact, from a practical standpoint but little relief can be offered in cases in which the animal is suffering from an internal neoplasm even if an accurate diagnosis is made; on the other hand, a considerable number of tumors affecting the exterior of the body are amenable to some form of treatment. It is in regard to this group of tumors that I would emphasize the need for a better understanding on the part of those called on to give an opinion as to the nature of what may be an innocent growth or a highly malignant one.

In recognizing malignant external tumors without the assistance of the microscope one should have in mind the parts most frequently involved by this type of tumor in the respective species. Since the most common external malignant tumors in most species are epithelial in nature (true carcinoma) a few examples will suffice to emphasize this point.

Tumors of the eye and its appendages in cattle and horses are practically always carcinomatous in nature, and tumors of the eye of dogs are usually adenomas and without the malignant possibilities of true carcinoma. Not infrequently fibromas occur in the skin of cattle, while many tumors of the skin of dogs are carcinomas or lymphosarcomas. Those of the anterior part of the mouth and of the lips of dogs are most likely to be papillomas, and those of the penis of the horse are usually carcinomas. Tumors of the mammae of bitches may be adenocarcinomas or adenomas, often with variable amounts of degenerative changes. Tumors of the mouth and sinuses of the head of the horse are usually carcinomas, while those of the vaginal vault of bitches are practically always transmissible lymphosarcomas. The frequently observed melanotic tumors of the horse and of the skin of swine are obviously so characteristic in appearance that they are probably the easiest of all tumors to diagnose. Occasionally, however, a non-pigment variety of this tumor is seen which is extremely malignant.

Although much can be determined concerning the true nature of a tumor by a careful study of its gross appearance, it is highly desirable, whenever possible, to remove a small piece of the growth for histologic examination. This can usually be done without pain to the animal, or if it is desired the removal can follow the injection of a small amount of procaine. If the tissue is placed immediately in 10 per cent formalin and forwarded to the laboratory, a diagnosis can usually be made within a few days at the most. When the exact nature of the tumor is determined its subsequent handling can then be outlined. In considering the prognosis of malignant tumors it is important to recognize that they exhibit many degrees or grades of malignancy. Some grow slowly with little or no tendency to invade the surrounding tissues, and others reveal a vigorous type of growth with early metastasis to adjacent lymph-nodes and organs. When metastasis has occurred, little can be done and one is justified in recommending the humane disposal of such an animal.

The early operative removal of a tumor of low-grade malignancy should always be attempted provided, of course, the value of the animal is such as to warrant the procedure. Even if the tumor is moderately malignant and if metastasis has not occurred, temporary relief is often possible by early treatment, and a valuable work animal or breeding animal may remain in

service several months longer than would have been possible without operative interference.

The very nature of the malignant growth should make one extremely cautious in offering an optimistic prognosis in any case of malignant tumor, but should not prevent one from attempting palliative measures, at least, in selected cases. Most of the benign types can be removed successfully if in operable situations, and many of those that are histologically malignant can, by operative procedures, be restrained from exerting a lethal effect for a considerable period, and occasionally a cure may be obtained.

Much of real value results from the treatment of certain malignant growths in human beings by the use of the Roentgen rays and radium, but these agents have been tried in only a few cases of neoplastic disease in animals. The work that has been done indicates that these agents are valuable aids in the treatment of selected cases. To those interested in this form of treatment of cancer, the observations of Little should be considered. For a period of several years Little[8] had the opportunity to study the effect of radium emanation on malignant growths of horses and dogs. In some instances complete cure was affected, the best results being obtained with a combination of irradiation and surgical procedures. Sarcomas and carcinomas responded to the treatment, and Little concluded that many of the malignant tumors of horses and dogs responded to radium emanation more readily than similar tumors of man.

Unfortunately relatively few veterinarians have access to the radium emanation tubes necessary for this form of treatment, but it is encouraging to know that radium is an agent capable, in many instances, of successfully combating and often destroying malignant cells.

In conclusion it seems proper to say that for the average practitioner the best chance for success in treating malignant growths is to detect them early in the course of the disease, determine their exact character by microscopic examination if possible, and if the site of the tumor is suitable for operation and metastasis has not occurred, removal by surgical means of all of the tumorous tissue. If all of the malignant cells are removed, the tumor will not recur and the result will be satisfactory.

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The A. V. M. A. will take charge of the City of the Quick and the Dead, August 13-16.

ADVERTISING A VETERINARIAN

The following editorial appeared recently in the Raleigh (N. Car.) *Times*, under the heading, "Dog Who Is Worth Keeping Deserves Hospital Care." It is so unusual that it is considered worthy of reproduction here. The veterinarian referred to is Dr. J. I. Neal, of Sanford, N. Car.

Since Louis Graves can defy the business department of the Chapel Hill Weekly by publishing a free advertisement of a competent veterinarian in his news columns, for once we will risk the same thing here by printing his story:

"Nancy, Dr. Mangum's handsome police dog, was taken ill recently. He made inquiries about dog hospitals and dog doctors, and the result of them was that he took his pet to Neal's Veterinary Hospital in Sanford. It is kept by a brother of W. B. Neal of Chapel Hill and has a reputation in many States.

"While he was on the street in Sanford, Dr. Mangum met a former pupil of his, Dr. Ludlow. When this young surgeon heard that Nancy was to have an operation he asked the privilege of assisting at it, and so he was present when the dog went under the knife.

"It was found that she had kidney trouble. After several days in the hospital she was brought back to Chapel Hill and placed under observation by a kidney expert of international fame, Dr. William de B. MacNider. She is getting better."

Naturally, Nancy has advantages not possessed by other dogs in her owner's professional occupation and associations, but the dog hospital is just another of the things that go to make life more complicated and what once appeared a liberal stipend look like merest chicken-feed. In fact, the Chapel Hill Weekly has in the past reported the happy issue of visits of expectant Chapel Hill dog mothers to canine maternity hospitals.

It is all, however, just a phase of the development of exactness in relation to any life worth saving. In old days even the best dogs became sick and died and passed away, to the regret, but little to the trouble of his master. Now the man who owns a dog considers that his treatment and its expense is a logical responsibility such as those he owes to his family.

What, if any, is the lesson? None, perhaps, except that a dog worth having is worth caring for and saving; and that the other kind remains an example of the carelessness that cheerfully lets its negligence endanger the things best loved, human and beast.

IMMUNIZATION OF CATTLE AGAINST BACILLARY HEMOGLOBINURIA

By LYMAN R. VAWTER *and* EDWARD RECORDS

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In 1914 an active investigation of a fatal unidentified disease of cattle was started by the Nevada Agricultural Experiment Station, ordinary clinical and laboratory methods having failed to establish a definite causative factor. Old newspaper files and reports by long-time residents seem to demonstrate beyond reasonable doubt that the disease under study had caused material death losses in western Nevada and adjacent portions of California for at least forty years.

Unfortunately many of these deaths were attributed to anthrax and anthrax vaccination was extensively practiced. As this procedure did not control the losses, anthrax vaccination was unjustly placed in bad repute.

At the beginning of the investigation, the disease was designated "hemorrhagic disease" on account of the striking hemorrhages found on the serous membranes and the large amounts of bloody fluid in the body cavities of cattle dead of this disease. Cattlemen and veterinarians termed the disease "red water" on account of the voiding of large amounts of dark red urine which is the outstanding clinical symptom. This term is recognized and widely used by stockmen and veterinarians when speaking of this disease of cattle common to the intermountain country of Nevada, southern Oregon, and California.

In our former publications the term ictero-hemoglobinuria has been used. As this term had been previously applied to a disease of sheep due to a different etiological factor, it now appears undesirable. We suggest, therefore, that the disease of cattle and sheep referred to in this paper be designated as bacillary hemoglobinuria.

Meyer,[1] Mack and Records,[2] and the writers[3] have recorded the clinical symptoms, postmortem lesions, epizoological observations and bacteriology. A sporulating anaerobic microörganism which we provisionally designated *Cl. hemolyticum bovis* and believed to be the cause of red water disease, was described by the present writers in a recent paper.[4] Since the publication of this

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paper, additional research has developed more evidence to support our statements. Artificial inoculation of cattle and rabbits with cultures of this anaerobe constantly produced clinical symptoms and postmortem lesions closely resembling the naturally occurring cases. The organism can be readily recovered from the carcasses of animals inoculated experimentally, thus fulfilling the established postulates of Koch.

The sporulating anaerobic bacillus previously described can be recovered from the liver infarct, blood and body organs in fresh cadavers of natural cases. Since the publication of our last paper, we have also repeatedly recovered this anaerobe from the bloodstream of living clinical cases of red water disease by special cultural methods.

Blood cultures from the affected cattle are made from the jugular vein under strict aseptic precautions, the blood being drawn direct into suitable anaerobic media sealed with hard petrolatum. We have had greater success in securing pure cultures in this manner than from the cadavers after death. Incidentally all but two cases showing positive blood culture died regardless of serum treatment.

Immunization Studies

With this evidence at hand, investigation was started to devise a method of routine immunization of cattle in areas where losses commonly occurred.

The bloody exudates found in the body cavities of cattle dead from natural infection, the lesion fluid at the site of inoculation in test cattle, and culture filtrates were examined for aggressins. The absence of aggressins in the body exudates and cultures was confirmed by the procedure of Scott.[5] Sterile culture filtrates and Mandler-filtered lesion fluid failed to activate washed suspensions of the organism sufficiently to kill susceptible test animals. Germ-free tissue exudates also failed to protect guinea pigs against subsequent injections of *Cl. hemolyticum bovis*.

Rabbits which had received either non-lethal doses of toxic cultures or liberal doses of non-toxic cultures proved immune when injected with lethal doses of young toxic cultures. This led to the belief that non-toxic cultures would be antigenic.

Mandler filtrates of 48-hour cultures produced sufficient immunity in about 75 per cent of rabbits and cattle to withstand at least 2 m. l. d. of toxic culture. However, the local reactions on animals injected experimentally were variable, following the

injection of different lots of filtrate prepared as nearly identically as possible.

Filtration apparently altered several lots of culture filtrate by removing some of the necessary antigenic substances, thus rendering the product variable in antigenic value.

In order to eliminate the variable nature of cultural filtrates, attention was directed toward developing an immunizing agent of greater uniformity. Six stock cultures of *Cl. hemolyticum bovis* which had been maintained for several years on beef-heart-peptic-digest media without added sodium chlorid or dextrose were selected for use in connection with such an investigation. These strains had almost, if not entirely, lost their sporulating tendency as a result of being carried as above. This was substantiated by the fact that cultures heated at 60° C. in a water-bath for one hour were killed. The cultures used were subjected to several successive passages through deep agar shakes to insure purity.

A large quantity of pig-stomach-liver digest[6] was prepared in order to insure a uniform culture medium throughout the work.

Sterilization of Cultures

A method of sterilizing whole culture suspensions of washed bacilli was sought which would cause the least decrease in antigenic values. Accordingly a study was made of the effect of heat and chemical agents upon whole cultures and suspensions of washed bacilli.

Peptic-digest broth pH 7.4, to which was added 0.2 per cent each of dextrose and dibasic potassium phosphate, was distributed in tall tubes 200x25 mm. and sterilized in the autoclave at eighteen pounds pressure for thirty minutes. Immediately following sterilization, sterile raw rabbit liver was added to each tube. The medium was then incubated for 48-hours to reveal any possibility of contamination incident to preparation. Young cultures sixteen to eighteen hours old were used for seeding these tubes.

The cultures were incubated for the desired period and then withdrawn from the tubes aseptically and subjected to low-speed centrifugation for five minutes to remove any meat débris present. The six cultures were then mixed and distributed in sterile rubber-stoppered vaccine vials.

Sterilization of the whole culture contained in vaccine vials was then attempted with heat or by the addition of chemicals. Immediately following the addition of chemicals or the exposure

to heat in a water-bath, the containers were mechanically agitated for three hours to break up clumps and thoroughly distribute the chemicals. The various samples were then incubated over night at 38° C., removed to the refrigerator the following day and stored at 5° C. until the sterility tests were made.

Washed bacilli from cultures of various ages were also suspended in solutions such as physiological salt solution, distilled water and 0.005 per cent NaOH to the volume of the original culture. These suspensions were also subjected to heating in a water-bath at 60° C. for one hour or the addition of chemicals.

Exposure to 60° C. in a water-bath for one hour completely sterilized the whole cultures and various suspensions of washed bacilli but such heating caused flocculating of the bacilli and complete clearing of the menstruum. Subsequent mechanical agitation produced only a temporary re-suspension. Clumping of bacilli frequently occurred when suspensions were subjected to heating at 50° C. for even thirty minutes, but this did not kill all of the organisms. Heat-treated whole cultures and washed suspensions were accordingly dismissed as unsatisfactory for further consideration.

Chemical Sterilization Satisfactory

The addition of 0.1 per cent formalin, or 0.5 per cent phenol, by volume, followed by mechanical agitation and over-night incubation, followed by storage at 5° C., efficiently sterilized both whole cultures and washed suspensions of organisms in seventy-two hours. Mechanical agitation and over-night incubation markedly accelerated the action of the germicidal agents and assured more dependable results. Chemical sterilization did not perceptibly alter the physical appearance nor cause clumping of the bacilli in whole cultures, but after two or three days partial sedimentation occurred, regardless of storage temperatures. This sediment, however, was readily broken up by manual shaking.

M'Ewen[7] reported the successful immunization of sheep with formalin-killed young cultures of vibrion septique, large quantities of culture being prepared by using the Waterman diffusion-shell method.

We modified M'Ewen's method and prepared several lots of antigen which were subsequently injected into rabbits. Peptic-digest broth pH 7.4, containing 0.2 per cent of dextrose and dibasic potassium phosphate, was used as a culture medium. In

place of the parchment diffusion-shells we substituted 50x150 mm. bags made of No. 60 cambric cloth. The broth was placed in Erlenmeyer flasks of the desired capacity. The cloth bags containing 25 grams of ground cooked beef heart were tied securely to prevent the escape of meat particles and then dropped into each flask and completely immersed in the broth. Sterilization was performed in the autoclave. Immediately following sterilization, the culture flasks were forcibly cooled in a water-bath to a temperature of 35 to 38° C.

Inoculation of the flasks was performed at once with young sixteen-hour cultures. This method produced a rapid growth of great density. Sealing with liquid petrolatum or the use of anaerobic jars was unnecessary, as growth was very active before appreciable amounts of oxygen were absorbed.

Optimum Incubation Period

In order to determine the period of incubation essential for the most potent antigen, cultures were subjected to chemical sterilization after 20, 48, 72 and 96 hours of incubation. Density readings were made on each sample with a Gates opacimeter[3] at the conclusion of the incubation period. Hydrogen-ion concentration was determined by the colorimetric method. The antigens were subjected to mechanical agitation, followed by incubation over night and removal to the refrigerator the following morning. Sterility tests were made 72 hours after the addition of the chemicals.

Lots 1, 2, 3 and 4 of antigen were prepared from avirulent cultures. Antigens 5A and 5B were prepared from the same strains, virulence being restored by repeated subculturing on beef-heart-peptic-digest media, containing 0.2 per cent ferric sulphate after the method of Scott.[9] This also restored sporulation which had to be again suppressed by several transfers on raw tissue media without added sodium chlorid or dextrose. Antigens 5A and 5B differed only in the germicide used.

Antigen 6 was prepared with avirulent cultures and was incubated for 48 hours but was not subjected to sterilization in any manner. In a former publication[4] we mentioned the presence of an unstable hemolytic toxin which persisted in unsealed cultures for 30 hours. Antigen 6 was purposely incubated until this toxin would be inactive. The variations in the preparation of the several lots of antigen are summarized in table I.

TABLE I—*Preparation of antigens*

ANTIGEN	AGE AND TYPE OF CULTURE	STERILIZING AGENT	DENSITY BY GATES METHOD	pH
1	20-hr. avirulent	Phenol 0.5%	1.1 cm.	5.6
2	48-hr. avirulent	Phenol 0.5%	1.1 cm.	5.8
3	72-hr. avirulent	Phenol 0.5%	1.1 cm.	6.0
4	96-hr. avirulent	Phenol 0.5%	1.4 cm.	6.0
5A	20-hr. virulent	Phenol 0.5%	1.1 cm.	5.5
5B	20-hr. virulent	Formalin 0.1%	1.1 cm.	5.5
6	48-hr. avirulent	Not sterilized	1.1 cm.	5.8

Rabbits and guinea pigs were injected with varying doses of each lot of antigen. In our experience guinea pigs behaved in an unsatisfactory manner and no definite conclusions could be made where they were used. Rabbits were found to be more suitable for immunity experiments.

Nine rabbits were injected subcutaneously with each antigen except No. 6. Three rabbits received 0.5 cc, three 1 cc, and three 2 cc each. In the case of antigen 6 only five rabbits were used. Local reactions followed at the point of injection, which varied somewhat according to the amount injected, consisting of an edematous swelling varying from 3 to 10 cm. in diameter. The local reactions subsided in 24 to 48 hours, without serious systemic disturbance. Antigens prepared from cultures incubated for 72 or 96 hours frequently failed to produce local reactions except in the largest doses given.

Antigen 5B, killed with 0.1 per cent formalin, failed to produce local reactions in six of the nine rabbits injected and they were scarcely perceptible in the remaining three. This feature is of interest and will be considered later.

IMMUNITY TESTS

Immunity tests were started seventeen days after injection on the rabbits receiving antigens 1 to 5B inclusive. At irregular intervals one individual from each dosage group was selected for test, the last test being made 48 days after the injection of antigen.

The m. l. d. of virulent toxic culture was determined in the usual manner and was found to be 0.25 cc for an 1800-gram rabbit. Two strains were used in the immunity test and the m. l. d. was approximately the same for both. Three control rabbits were injected with virulent toxic culture whenever an immunity test was made on treated rabbits. The controls died without exception in 16 to 36 hours.

Typical lesions, including hemoglobinuria, were observed in every control. The inoculated organism could be recovered from the thigh lesion and heart-blood. Table II gives a summary of the results obtained.

TABLE II—*Immunity tests in rabbits*

ANTIGEN	RABBITS INJECTED	DIED FROM INTERCURRENT CAUSES	RABBITS TESTED	DIED	SURVIVED IMMUNITY TESTS	
					NUMBER	%
1	9	1	8	1	7	87.5
2	9	0	9	3	6	66.6
3	9	0	9	3	6	66.6
4	9	0	9	5	4	44.4
5A	9	0	9	2	7	77.0
5B	9	0	9	7	2	22.0
6	5	0	5	0	5	100.0

Antigen 1, which was prepared from young toxic cultures, showed the highest percentage of survivors of all the killed antigens.

Antigens 2 and 3 show the same percentage of survivors but less than antigen 1.

Antigen 4 had only 44 per cent survivors.

Antigen 5A, which was essentially the same as antigen 1, excepting that it was prepared with virulent cultures, protected 77 per cent of the rabbits. One rabbit which died following the injection of virulent culture did not show lesions characteristic of this infection but did show a severe coccidiosis infection. We did not attribute the death of this individual to the test inoculation.

Antigen 5B was killed by formalin to compare the effect of formalin and phenol on the antigenic properties.

As previously stated, formalin-killed antigens did not produce the local reactions characteristic of phenol-killed antigens or living cultures.

Immunity test shows only 22 per cent survivors among the formalin-killed antigen group. In our earlier work in connection with Mandler filtrates and cultures it appeared that immunity was probably produced by toxoids. Whatever the antigenic substance is, formalin apparently destroys it to a large extent.

Antigen 6 was a viable but non-toxic culture which has been held in the refrigerator at 5° C. for five days after the completion of the incubation period.

In this group the rabbit dosage ranged from 0.1 to 1 cc. The local reactions incident to injection were comparable to those observed following the injection of killed antigens. Immunity test was made twenty days after injection with live culture. All of this group survived.

It appears from the foregoing results that young phenolized antigens incubated 20 to 24 hours are most desirable excepting perhaps the living non-toxic cultures. Furthermore, it appears that living cultures can be used without danger, providing the incubation period was long enough to insure the inactivity of the hemolytic toxin produced by this organism.

The fairly uniform results obtained on laboratory animals led us to try phenol-killed young cultures on cattle kept on heavily infected premises during the summer seasons of 1926 and 1927.

Preparation of Bacterin

The following procedure has been adopted for the preparation of bacterin on a large scale at this Station:

Stock cultures are transferred once a month on beef-heart-peptic-digest media[6] containing 0.2 per cent each of dextrose and dibasic potassium phosphate. Subcultures are incubated 48 hours and then stored in the refrigerator at 5° C. Sporulation occurs in media containing added sodium chlorid and dextrose but does not readily occur when cultures are repeatedly subcultured on raw tissue media without the addition of same.

Stock cultures must be examined at frequent intervals to exclude contamination. Prior to the preparation of a lot of bacterin, stock cultures are transferred twice a day for three or four days on raw rabbit-liver-peptic-digest media without added sodium chlorid or dextrose and sealed with petrolatum. Growth is very rapid in this medium and consists entirely of vegetative bacilli.

Twelve to sixteen-hour cultures grown on raw tissue media are used for seeding media intended for the preparation of bacterin. The medium used for preparing large quantities of bacterin is simple, namely:

Peptic digest broth	pH 7.4
Dextrose (c.p.)	0.2%
Dibasic potassium phosphate	0.2%

Eight hundred and fifty cc of media is placed in each liter Erlenmeyer flask. Twenty-five grams of cooked ground beef heart, enclosed in a No. 60 cambric cloth sack, is added to each

flask and completely submerged in the broth. The sacks are securely tied to prevent escape of meat particles.

Sterilization is performed in the autoclave at eighteen pounds pressure for 45 minutes. Immediately following sterilization the flasks are quickly cooled in a water-bath to a temperature of 35 to 38° C.

Seeding of the flasks is performed immediately after cooling. We use not less than five strains of the anaerobe designated *Cl. hemolyticum bovis* for each lot of bacterin, but each flask is seeded with only one strain. The culture flasks are incubated for twenty hours at 38° C., which permits the cultures to attain a maximum density.

A Gram stain is made from each flask to exclude the presence of contamination. The majority of the bacilli should be Gram-negative but Gram-positive forms are common at this time. Spores should not be present.

Density readings are made with a Gates opacimeter[8] at the conclusion of the incubation period. In fifteen lots of bacterin the density averaged 1.1 cm.

Readings made with colorimetric methods at the conclusion of twenty hours incubation in eight lots of bacterin showed a pH range from 5.6 to 6.2. Recent lots of bacterin have been titrated with a potentiometer and checked very closely with the colorimetric method.

The contents of the flasks are aspirated aseptically into a large sterile bottle. Five per cent phenol in distilled water is added to make 0.5 per cent phenol by volume. The phenolized culture is vigorously agitated and permitted to stand at room temperature for one to two hours. Bottling is performed into the final containers which are closed with rubber-diaphragm stoppers.

The final containers are mechanically agitated for one hour to distribute the phenol thoroughly, incubated over night at 38° C., then removed to the refrigerator the following morning. Sterility test is not conducted earlier than 72 hours after the addition of phenol.

The sterility test includes media suitable for both aerobes and anaerobes. Sterility test cultures are incubated 72 hours. At the completion of a satisfactory sterility test, a group of rabbits is injected with doses of the bacterin ranging from 1 to 5 cc. Rabbits have always survived this test with the occurrence of local swellings at the site of injection ranging from 2 to 8 cm. in diameter, which may vary in proportion to the size of the dose.

The local reaction persists for twenty-four to thirty-six hours, then gradually subsides. Occasionally some of the rabbits may be "off feed" the day following injection. Upon completion of satisfactory sterility and laboratory animal tests, the bacterin has been used for the injection of cattle.

Field Trials on Cattle

The fact that red water disease may cause heavy loss on certain premises for one season and then virtually disappear for a period of years has been repeatedly observed. This feature makes it difficult to draw conclusions from immunization experiments unless a large number of cattle are included on a fair number of premises in an infected district.

One ranch used as a summer range for seven or eight hundred cattle had a history of seventy deaths from red water disease in 1921. A somewhat lighter loss occurred in 1924 and 1925, which were exceptionally dry years, and accordingly unfavorable for a high incidence of red water disease. We secured this group of privately-owned cattle and a number of others under a co-operative agreement for the experimental trial of red water bacterin during the years 1926 and 1927. During these years irrigation and natural water was more abundant and the case incidence greater.

The cattle injected with bacterin were identified by ear-punching. Five cc of bacterin was injected subcutaneously. All classes and ages of cattle excepting suckling calves were included in the experiment. Systemic disturbances did not occur subsequent to injection of the bacterin. Occasionally a slight local reaction was noticed in three to six hours after injection of bacterin but subsided entirely the next day. A total of 3828 cattle were injected with bacterin during 1926 and 1927. The results are shown in tables III and IV.

The experimental results recorded in table III show ten suspect cases among the treated animals. Owing to a written agreement with the owners, we were obliged to examine and treat all suspect and actual cases of this disease which occurred in the cattle covered by the agreement. Constant loss from this disease in former years caused considerable alarm to owners whenever sick cattle were found, regardless of the cause. None of the cases classed as suspects among the treated animals developed definite symptoms of red water disease.

TABLE III—*Cattle injected with 5-cc doses of bacterin during 1926*

	NUMBER	PERCENTAGE
Total treated animals	1636	82.3
Total positive cases of red water among treated animals	0	0.0
Total doubtful cases of red water among treated animals	10	0.6
Total positive and doubtful cases of red water among treated animals	10	0.6
Total control animals	354	17.7
Total positive cases of red water among control animals	4	1.13
Total doubtful cases of red water among control animals	4	1.13
Total positive and doubtful cases of red water among control animals	8	2.26

Four suspect cases were observed among the untreated controls; however, four deaths also occurred among the controls from red water disease, which were positively confirmed by clinical or necropsy examination.

Digestive disturbances were accountable for a number of suspect cases. The incidence of suspect and actual cases was over three and one-half times greater in the untreated than in the treated animals.

TABLE IV—*Cattle injected with 5-cc doses of bacterin during 1927*

	NUMBER	PERCENTAGE
Total treated animals	2192	80.6
Total positive cases of red water among treated animals	4	0.18
Total doubtful cases of red water among treated animals	6	0.27
Total positive and doubtful cases of red water among treated animals	10	0.45
Total control animals	525	19.4
Total positive cases of red water among control animals	11	2.09
Total doubtful cases of red water among control animals	3	0.57
Total positive and doubtful cases of red water among control animals	14	2.66

The experimental results recorded in table IV for the year 1927 cover a larger number of animals than shown for 1926.

Four deaths occurred from red water disease among the treated animals. One death occurred in a large group of range cattle two months after vaccination. No further loss occurred in this group until five and one-half months after vaccination. This group of

cattle is wintered on badly infected premises with warm springs and losses started again in December. This group was again immunized and went through the winter and early spring without further death loss. Losses started again in July, 1928.

The incidence of positive cases among the untreated animals was higher in 1927 than it was in 1926.

In 1927 the incidence of actual cases among the treated animals was 0.18 per cent, whereas among the untreated controls the incidence was 2.09 per cent, or slightly more than eleven times greater than in the treated group.

The results attained with bacterin indicated that immunity probably does not last longer than five or six months. This is probably sufficient in most cases to carry cattle through the summer and autumn seasons when the disease is most prevalent. Very few cases occur during the winter months.

This product was released in 1928 for general use in Nevada by graduate veterinarians.

There is the possibility that bacterin may not confer sufficient immunity in a few instances to protect cattle kept on heavily infected premises throughout the year. Furthermore, some areas harbor infection of a much greater virulence than others. In anticipation of the future, investigation was continued toward the development of an immunizing agent to meet such contingencies.

During the summer season of 1928, further work was done with a group of 740 cattle which are kept throughout the year on badly infected premises. This group ordinarily has losses due to red water disease during the winter months. In an attempt to establish a more prolonged immunity, a live glycerinated vaccine was used on this group in June, 1928. The fact that rabbits would tolerate 5-cc doses of non-toxic cultures has already been mentioned in this paper. Further investigation revealed that rabbits would tolerate as high as 10 cc of glycerinated non-toxic culture without serious disturbance. Glycerin in concentrations of ten to forty per cent by volume kept cultures viable for at least ninety days.

The method of preparing glycerinated live vaccine was similar to that described in this paper for the preparation of bacterin. Cultures were incubated 26 hours. At the completion of the incubation period the cultures were aspirated into a large sterile bottle and 25 per cent sterile glycerin by volume was added. The

glycerinated culture was stored for one week at 5° C. prior to the preliminary animal tests.

Rabbits and cattle were injected with doses ranging from 2 to 10 cc. The rabbits developed large local reactions at the site of injection and considerable systemic shock but no deaths occurred.

The preliminary test group of cattle consisted of five pregnant cows and five yearlings of both sexes. Two of the cows had been treated in 1926 and 1927 with red water bacterin produced by this Station. The remainder of the test group had never received any form of red water immunization prior to this test. Doses ranging from 2 to 10 cc were given subcutaneously.

Definite local reactions appeared in six hours after the injection of this live glycerinated vaccine. The local reactions were most pronounced in the yearlings, which received 8 and 10-cc doses and consisted of a local swelling approximately 10 cm. in diameter. The local reactions subsided entirely in 24 hours. No further reaction of any kind occurred during a five-day observation period. A 5-cc dose of live glycerinated vaccine therefore appeared to be reasonable and safe.

The herd was divided into two groups. The 622 cattle making up group I received 5 cc of glycerinated live vaccine subcutaneously. They were identified by ear-punching. The 118 cattle in group II were not treated nor had they received any form of red water immunization at any previous time. They served as controls and were not ear-marked.

Approximately eight months have elapsed since vaccination and the herd has been kept constantly on badly infected pastures and fields. To date the incidence of red water disease has been 3.4 per cent among the unvaccinated controls and none among the vaccinated animals. Final conclusions regarding the value of live glycerinated vaccine will not be made until June 1, 1929, when the experiment on this herd will be terminated.

Summary

1. The role *Cl. hemolyticum bovis* plays as the direct causative factor or red water disease or bacillary hemoglobinuria has been confirmed.

2. The antigenic properties of *Cl. hemolyticum bovis*, have been demonstrated.

3. A method of preparing whole culture bacterins and glycerinated culture vaccines with *Cl. hemolyticum bovis* is described.

4. A total of 1636 head of cattle were given 5-cc doses of phenol-killed bacterin in 1926. The percentage of positive and suspect cases of red water among 354 head of controls was over three and one-half times that among the untreated cattle.

5. A total of 2192 head of cattle were given 5-cc doses of phenol-killed bacterin in 1927. The percentage of positive cases of red water disease among 525 head of controls was over eleven times that among the treated.

6. In 1928 over 4000 head of cattle were given 5-cc doses of phenol-killed bacterin as a routine procedure by graduate veterinarians. No bad results were noted and protection appeared to be complete for at least six months.

7. A group of 622 head of cattle were given 5-cc doses of living non-toxic cultures of *Cl. hemolyticum bovis*, plus 25 per cent glycerin, under experimental conditions in 1928. There were no bad effects. Protection has been complete for eight months. Four deaths from red water occurred among 118 head of controls during the same period.

CONCLUSIONS

1. Both phenol-killed whole-culture bacterins and glycerinated non-toxic live-culture vaccines prepared from *Cl. hemolyticum bovis* are harmless and efficient antigenic agents for the scientific control of red water disease or bacillary hemoglobinuria.

2. The glycerinated non-toxic culture vaccine appears to confer a more solid and lasting immunity.

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Erratum

In the paper, entitled, "Studies with Bovine Skin Lesions," by Elder, Lee and Phelps, published in the July issue of the JOURNAL reference 10 as given is incorrect. It should be as follows:

[10]Mitchell, Chas. H.: Bovine subcutaneous tuberculosis. Jour. A. V. M. A., lxxiii (1928), n. s. 26 (4), pp. 493-506.

The Fourth World Poultry Congress will be held in Crystal Palace, London, England, July 22-30, 1930.

REPORT OF EXPERIMENTAL WORK IN THE CONTROL OF BOVINE INFECTIOUS ABORTION*

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We reported[1] in 1926, the results of two years' investigations in the control of bovine infectious abortion. The investigations were continued and the results obtained over a five-year period are now presented.

This experiment was undertaken to obtain data on the spread of bovine infectious abortion, or Bang's disease. M'Fadyean seems to have been the first to have suggested and put in opera-

FIG. 1. The infected herd is kept in the barn and paddock at left, the clean herd on the right. This also shows the difference in the levels of the two barns.

tion a method of control based on the establishment of herds free of Bang abortion disease by the aid of the serum tests. In this country, Barnes,[3,4] Simms and Miller,[5,6] Newsom and Cross,[7] and Rettger[8,9] and his associates have published results of investigations indicating that it is possible and practicable to control Bang abortion disease in this manner.

Two herds of cattle were established by the Veterinary Division at University Farm. They are kept in two barns, separated by not more than 75 feet (see fig. 1). The herds are attended by the

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†R. E. Lubbehusen, formerly a member of the Staff of the Division of Veterinary Medicine, University Farm, aided in securing some of the data here reported.

same men. One herd is infected with *Bact. abortus* and the other herd is free of the germ. The infected herd (fig. 2) is maintained at about ten animals and the clean herd (fig. 3) at about twenty animals. The blood of the cattle is tested for antibodies for *Bact. abortus* each month. Further details are given in a previous article.[1]

During the past three years, additional information has been presented to indicate the importance of Bang's disease and the need of hastening its control. Additional data[10,11,12] have been presented to show that immunological agents will not satisfactorily solve the problem presented by this infection. There is no question that the injection of living vaccines will reduce the

FIG. 2. The infected herd.

number of abortions occurring in a badly infected herd. We pointed out this fact from the results of our own investigations.[13] The use of these products, however, is attended with many difficulties. The most important is that the occurrence of abortions, resulting from the Bang organism, cannot be eliminated nor in most cases reduced to the desirable minimum. Sterility or failure to breed has been noted, by some investigators, to follow the use of these products. The occurrence of cases of undulant fever in man, and their possible incidence from infected milk, has brought to our attention, more forcibly than ever before, the danger of producing spreaders by the use of vaccines. Finally there will always be an infected herd and the use of the vaccines must be continued from year to year.

The advantages of a herd free of Bang's disease, from an economic standpoint, are well illustrated by the published results from the Storrs Experiment Station, and from data presented by the Nebraska Station. The Pennsylvania plan of keeping herds free of the disease has been adopted by other states. The live stock sanitary authorities of Pennsylvania have made Bang's disease reportable and quarantinable. New Jersey has a law which enables owners of infected animals to obtain a herd free of the infection under the guidance of the state live stock sanitary authorities. The desire of owners to have their herds free of the disease is manifested by the greatly increased number of blood tests for the presence of infection which all laboratories doing

FIG. 3. The clean herd.

this work have been called on to make, during the past two years. Cattle-breeders are becoming aware of the dangers and losses from this infection and are urgently requesting information about the disease and aid in reducing its losses.

In a very recent publication, Rettger and his associates[8] have reported data on the detailed control of Bang's disease in four herds, on the basis of the blood test and the segregation or disposal of reactors and the building up of negative herds. One of the conclusions from their work is: "Elimination of the Bang abortion disease results in an improved reproductive condition of the herd which in itself is of supreme economic importance. This, added to the value of a greatly increased milk supply may,

in the course of only a short period, turn a loss account into one of real profit."

In abortion control, we are constantly confronted with what to do with the infected animal. In tuberculosis control work, the status of the diseased bovine has been settled as far as this country is concerned. All infected animals are slaughtered. This system has been forced by the failure of the so-called "Bang system" of tuberculosis control. Even in countries outside the United States, where the segregation method for tuberculosis elimination has been carefully carried out for many years, the disease continued to be a real scourge. This country is far ahead in its control of bovine tuberculosis. Biological products have

FIG. 4. A view from the back of both barns. Cattle in the infected herd are seen at the right and those of the clean herd are seen on the left.

not yet been proven effective in stopping the ravages of bovine tuberculosis.

What then, are we going to advise the owner to do with the animal infected with *Bact. abortus?* To obtain an answer to this question and to procure information on methods of spread of the disease this experiment was initiated. The proximity of the two herds is indicated in fig. 1. Particular attention is called to the drainage conditions. There is a drop of over six feet from the barn holding the infected herd to that housing the clean herd. Considerable surface water flows from around the barn containing the infected herd past the barn containing the clean herd, especially in the spring of the year and following severe

rains. During the five years, six different men have been employed to care for the animals. They were ordinary farm laborers. No special precautions, such as dipping the feet in disinfectant, are taken to prevent carrying the germs.

Three Cases of Infection in Clean Herd

During the progress of the experiment, three cases of infection with the Bang organism have occurred in the clean herd. The first infection was in animal 51. This individual was purchased January 11, 1924, and was approximately eleven months old. At that time blood tests were negative and she was placed in the clean herd. She was bred three times and conceived on the May 4, 1924, service. The complement-fixation test was positive on May 1, 1924; agglutination, negative. The agglutination test became positive on September 1, 1924, and she was transferred to the infected herd, September 6, 1924. She calved February 5, 1925. *Bact. abortus* was not isolated from placenta or colostrum. Complement fixation remained positive, agglutination became negative. She calved the second time, January 2, 1926. *Bact. abortus* was isolated from the placenta. This was the only time the organism was isolated from this animal. Repeated examinations of colostrum, milk, placenta and uterine exudate were always negative. The agglutination and complement-fixation tests became negative and remained so until October 1, 1926, when both became positive. She calved the third time, November 10, 1926. All bacteriological examinations were negative for *Bact. abortus*. The animal developed a slight metritis, although the placenta was discharged promptly. During treatment in November, 1926, a soft-rubber horse-catheter used in irrigating the uterine cavity punctured the wall of the uterus and over a gallon of fluid flowed into the peritoneal cavity. The animal made a nice recovery, but developed adhesions of the uterine cornua and an abscess of the right ovary. She was condemned as permanently sterile and sold February 2, 1927, for slaughter.

The second animal to become infected in the clean herd was No. 22, calved June 10, 1922. She was in the clean herd until July 1, 1925. Her dam was a positive reactor. *Bact. abortus* was isolated from the milk of the dam during the time animal 22 was allowed to nurse. The serum tests after the first few months were negative, however, until July 1, 1925. She first calved August 20, 1924, and the placenta was retained. A bacteriological exami-

nation of the uterine discharge on August 29, 1924, showed *Staph. albus*. Involution of the uterus progressed rapidly and she conceived on the first service, October 27, 1924. Both serum tests, June 1, 1925, were negative. The tests on July 1, 1925, were positive. She was immediately removed from the clean herd and placed in quarantine. Another test, July 20, also was positive. She calved July 24, 1925, and expelled her fetal membranes within four hours. *Bact. abortus* was isolated from the placenta and from the colostrum. Involution of the uterus was retarded as a result of endometritis. She was placed in the infected herd on August 10, 1925. She calved the third time, September 3, 1926. Bacteriological examination of the placenta was negative for *Bact. abortus*. The colostrum was positive. Her fourth calf was dropped, August 20, 1927, and her fifth calf, December 20, 1928. *Bact. abortus* was not present. *Staph. albus* was found in the uterine discharge which developed following both calvings. The abortion germ is found consistently in her milk. This animal is an illustration of what is usually called an "immune carrier."

No. 57 was the third and last animal to become infected in the clean herd. Born September 18, 1924. Dam was No. 21. Bred the second time, October 25, 1925, and conceived. Blood tests negative until April 1, 1926, when both were positive and she was transferred to the infected herd, April 3, 1926. She aborted, June 10, 1926. *Bact. abortus* was isolated from placenta and colostrum. This cow calved again, June 24, 1927. *Bact. abortus* was again isolated from the placenta and colostrum. Calf was weak and died in ten hours. She calved the third time, June 1, 1928. Calf was weak but able to stand. The cow stepped on her calf and the autopsy revealed a ruptured liver. Bacteriological examination of placenta was negative, colostrum positive, and of the calf negative. She was bred again and is due to calve, April 15, 1929. Serum tests have been consistently positive. *Bact. abortus* has been isolated repeatedly from the milk.

The Infected Herd

Animal 62 came into the infected herd, January 16, 1925. She was born December 21, 1920. She had aborted twice and had produced one healthy calf before coming into the herd. Since being in the herd she has aborted five times. Usually she has conceived promptly after aborting. Following the last two, she developed cystic ovaries and it was necessary to treat her many times before conception took place. This animal has the unusual

record of seven abortions out of eight pregnancies. *Bact. abortus* is regularly isolated from the fetus, colostrum and milk. The serum tests are consistently positive.

Animal 63, a heifer, was calved January 7, 1925. Her dam was No. 47. She was kept in the infected herd and the serum tests became negative. She was bred, November 9, 1925. Conceived on the first service. Serum tests positive, May 1, 1926. Calved August 1, 1926. (This calf is No. 80 of the clean herd.) *Bact. abortus* was isolated from the placenta and colostrum. This animal was bred seven times but failed to conceive. No pathological changes of the genitalia could be detected on physical examination. She was sold for slaughter, September 2, 1927. This animal is an example of one which will not conceive even in the absence of any clinically observed pathology. We have slaughtered and examined carefully, pathologically and bacteriologically, the genitalia and other organs of similar sterile cows without finding changes which, in our minds, were sufficient to account for the failure to conceive.

The pathogenicity of the infection of the herd was tested in the fall of 1927 and winter of 1928. Nearly 18 months had passed since any Bang abortion-infected animals had been found in the clean herd. It was known, of course, that the germ existed in the infected herd but its ability to produce disease in clean cattle was undetermined. A lack of pathogenicity would account for the failure of the disease to be transferred under the conditions of the experiment.

On August 24, 1927, two animals (74 and 77) were transferred from the clean herd to the infected one and on September 10, 1927, a third animal (83).

No. 74 was calved January 2, 1926. Her dam was No. 51, an infected individual. No. 74 was placed in the clean herd as a young calf and always had negative serum tests. She was bred first, February 19, 1927, and did not conceive. Bred the second time, May 24, 1927, and pronounced in calf, July 7, 1927. She remained negative until the January 3, 1928, test, at which time she was strongly positive. She calved a live but premature calf, February 2, 1928. The calf died on February 5, 1928, and *Bact. abortus* was isolated from the lungs. *Bact. abortus* was also isolated from the colostrum. It was unfortunate the guinea pigs inoculated with the placenta died the following day. Cultures made directly from the placenta were negative for the Bang germ. Eight services and repeated treatments were necessary to induce

the second pregnancy. She is now in calf from the October 18, 1928, service. *Bact. abortus* has been isolated from her milk.

No. 77 was calved March 29, 1926. Her dam was No. 35 of the clean herd. She was bred first, May 5, 1927, and pronounced in calf, July 7, 1927. She remained negative to the blood tests until January 3, 1928, when she became strongly positive. She calved January 21, 1928. There was an excessive brownish-colored discharge, and *Bact. abortus* was isolated from the placenta which was retained. Only enough of the membranes could be removed for a bacteriological examination. Her calf became negative to the blood tests and has remained a healthy individual to this date. The metritis became more severe, and she did not conceive until the August 3, 1928, service. She still remains pregnant. *Bact. abortus* has been isolated from her milk.

No. 83 was calved October 15, 1926. Her dam was No. 48 of the clean herd. No. 83 was negative to the serum tests and was bred first to bull 93 of the infected herd on September 14, 1927, and conceived. She remained negative to the serum tests until April 2, 1928, when she became strongly positive. She aborted May 16, 1928. *Bact. abortus* was isolated from the placenta and colostrum. Her genital organs returned to normal rather quickly, and she was bred first, June 15, 1928. She did not conceive until the fourth service on September 13, 1928. She is now in calf. Serum tests in all three cases (74, 77 and 83) remain strongly positive.

This test showed the high pathogenicity of the Bang germs in the infected herd. It is remarkable that *all three* clean animals should become infected during their first pregnancy in which they were in contact with the infection. It is true, of course, that they were of the most susceptible group, heifers in their first pregnancy.

No. 88 calved July 16, 1927. Her dam is No. 47. This animal has remained positive to the serum tests from birth. She was bred, May 26, 1928, conceived on the first service and is now in calf.

During the five years this experiment has been in progress, three abortions have occurred in the clean herd. The first was No. 48. This animal was purchased, December 21, 1923. She was approximately 8-9 years old at this time. No previous history known. Serum negative to the blood tests. Bred first, December 29, 1923, and again, March 9, 1924. She conceived by this latter service and aborted a male fetus, September 30, 1924. A mucor

was isolated from the placenta and from the stomach contents of the fetus. This organism agreed culturally and morphologically with the one described by Smith.[14] The animal retained a portion of her placenta and developed cystic changes in one ovary, but nevertheless made a fairly rapid recovery. She was bred, October 27, 1924, December 20, 1924, and February 19, 1925. She conceived by this last service and calved, November 22, 1925. No evidence of infection could be found in the placenta which was expelled within four hours following parturition. She was rebred, January 13, 1926, and conceived by the first service and calved, October 15, 1926, a heifer calf (83). Placenta was expelled within four hours. No evidence of infection found on bacteriological examination. Cow not rebred, but sold for slaughter, February 2, 1927. Serum tests always negative for Bang infection.

The second abortion was in animal 26. She was calved September 7, 1922. Bred first, December 28, 1923, and conceived by the first service. Calved on September 28, 1924. Calved the second time, September 10, 1925, and her third calf was born, July 24, 1926. Parturitions were all normal and placenta readily expelled. She was rebred, September 11, 1926, and pronounced in calf, October 20, 1926. On November 16, 1926, a small embryo was found in the vagina.

Bacteriological examinations of the embryo and uterine discharges failed to show any specific organism. Bred on February 9, 1927, and conceived by the first service. Calved normally, November 14, 1927. Bacteriological examination of products of parturition negative. Bred on January 3, 1928, conceived and was sold for slaughter, June 6, 1928. Serum reactions always negative. Milk and colostrum always negative for *Bact. abortus*.

The third animal to abort was No. 66. She was born May 15, 1925. Bred first, June 3, 1926, and was pronounced in calf, July 26, 1926. Aborted on January 25, 1927. *Aspergillus fumigatus* was isolated in pure culture from the stomach of the fetus and from the uterine discharge. Placenta retained. No evidence of *Bact. abortus* found. Bred twice, last on June 1, 1927, conceived and calved normally, March 4, 1928. Bacteriological examinations negative. Placenta expelled promptly. Bred on May 4, 1928, and conceived by the first service. Is now in calf. Serum reactions always negative for *Bact. abortus* antibodies.

We desire to point out that these three animals were left in the clean herd. Nothing more than ordinary stable hygiene was ob-

TABLE I—*Data on clean herd, January 1, 1924, to January 1, 1929*

Cow	Age	Services	Living Calves	Abortions	Dead Calves	Retained Placentas	Present Breeding Condition	Remarks
6	6 yrs.	9	4	0	0	0	In calf at time of sale	Sold for slaughter, Oct. 28, 1926
21	6 yrs. 6 mos.	9	4	1?	0	0	In calf	
26	6 yrs. 3 mos.	6	4	1	0	0	In calf	Sold for slaughter, June 6, 1928. Abortion due to undetermined cause
30	4 yrs. 2 mos.	4	2	0	0	0	In calf	Sold for slaughter, Dec. 16, 1926
34	3 yrs. 11 mos.	5	2	0	0	0	In calf	Sold for slaughter, Dec. 6, 1926
35	4 yrs.	9	3	0	0	0	In calf	Sold for slaughter, Oct. 28, 1926
39	2 yrs.	1	1	0	0	0	Not bred at time of death	Died suddenly of cerebral hemorrhage, Dec. 27, 1924
40	5 yrs. 6 mos.	5	4	0	0	0	In calf	
42	2 yrs.	3	1	0	0	0	Not bred	Killed, May 19, 1925. Bronchial pneumonia and pulmonary emphysema
43	3 yrs. 2 mos.	6	2	0	0	0	In calf	Sold for slaughter, April 1, 1926
44	8 yrs.	12	0	0	0	0		Sterile. Killed, Oct. 6, 1925
45	4 yrs.	6	1	0	0	0	In calf	Sold for slaughter, April 1, 1926
48	11 yrs.	5	2	1	0	0	Not bred	Sold for slaughter, February 2, 1926. Abortion associated with a mucor
49	5 yrs.	6	4	0	0	0	In calf	
55	4 yrs. 3 mos.	4	3	0	0	1	In calf	
59	4 yrs. 2 mos.	7	3	0	0	0	In calf	
60	4 yrs. 11 mos.	6	1	0	1	1	In calf	
64	3 yrs. 10 mos.	5	2	0	0	1	In calf	
66	3 yrs. 6 mos.	4	1	1	0	1	In calf	Abortion associated with *Aspergillus fumigatus*
68	2 yrs. 11 mos.	4	2	0	0	0	In calf	
69	1 yr. 6 mos.	5	0	0	0	0		Sold for slaughter, Feb. 2, 1927. Sterile

TABLE I—*Data on clean herd, January 1, 1924, to January 1, 1929—Continued*

Cow	Age	Services	Living Calves	Abortions	Dead Calves	Retained Placentas	Present Breeding Condition	Remarks
70	3 yrs. 2 mos.	3	2	0	0	0	Open	
71	3 yrs.	3	2	0	0	0	In calf	
73	3 yrs.	2	2	0	0	0	Open	
75	2 yrs. 9 mos.	2	1	0	1	0	Open	
78	2 yrs. 6 mos.	3	0	0	1	0	Open	
79	2 yrs. 5 mos.	2	1	0	0	0	In calf	
80	2 yrs. 4 mos.	2	1	0	0	0	Open	
81	2 yrs. 3 mos.	3	0	0	1	0	In calf	
82 22	2 yrs. 3 mos.	2	1	0	0	0	In calf	
86	1 yr. 5 mos.	2	0	0	0	0	In calf	
87	1 yr. 3 mos.	2	0	0	0	0	In calf	
89	1 yr. 4 mos.	1	0	0	0	0	In calf	
111	Bull							Sold for slaughter, April 1, 1926
222	Bull							Sold for slaughter, May 17, 1927
270	Bull							Now being used
878	Bull							Sold for slaughter, March 25, 1924

served in their care. In no case did these non-specific abortions spread in the herd.

Tables I and II will give some detailed information in regard to the different animals used in the experiment. Space will not permit a detailed history of each individual, although such is available. It will be noted that animals were sold from time to time. The funds supporting this work would permit the maintenance of but a limited number of animals (approximately 20 head in the clean herd and 10 head in the infected herd). The older animals from the clean herd were sold off. Their places

TABLE II—*Data on infected herd, January 1, 1924, to January 1, 1929*

COW	AGE	SERVICES	LIVING CALVES	ABORTIONS	DEAD CALVES	RETAINED PLACENTAS	PRESENT BREEDING CONDITION	REMARKS
13	4 yrs.	2	1	0	0	1		Slaughtered, Oct. 30, 1924. Sterile. Large abscess involving uterus and ovaries
22	6 yrs. 6 mos.	8	5	0	0	2	Open	Transferred to infected herd, August 10, 1925
46	3 yrs. 9 mos.	7	2	0	0	2	In calf	Sold for slaughter, Oct. 28, 1926
47	6 yrs. 6 mos.	9	3	2	0	3	Not in calf	Killed, June 11, 1928. Multiple abscesses of liver, massive adhesions
50	7 yrs.	6	3	1	0	0	Not in calf	Sold for slaughter, Feb. 1, 1928
51	4 yrs.	5	3	0	0	1	Not in calf	Sold for slaughter, Feb. 2, 1927. Sterile. Abscess of right ovary and oviduct adhesions. Transferred to infected herd, Sept. 6, 1924
54	8 yrs.	4	0	1	0	1	Not in calf	Sold for slaughter, Dec. 28, 1925
57	4 yrs. 3 mos.	6	1 (died the same day)	2	0	2	In calf	Transferred to infected herd, April 3, 1926
62	8 yrs.	18	1	7	0	3	Open	
63	2 yrs. 8 mos.	7	1	0	0	1	Sterile	Sold for slaughter because sterile, Sept. 21, 1927
65	4 yrs.	11	0	1	0	1	Sterile	Animal purchased at time of abortion. Slaughtered because of abscesses of uterus, May 21, 1925
67	5 yrs.	12	0	1	0	0	Sterile	Animal purchased, August 5, 1925. Aborted same day. Sold for slaughter, Oct. 28, 1926, because sterile
74	3 yrs.	10	0	1	0	0	In calf	Placed in herd, Aug. 24, 1927
77	2 yrs. 8 mos.	3	1	0	0	1	In calf	Premature calf. *Bact. abortus* isolated from placenta. Placed in herd, August 24, 1927
83	2 yrs. 2 mos.	4	0	1	0	0	In calf	Placed in herd, Sept. 10, 1927
88	1 yr. 7 mos.	1	0	0	0	0	In calf	In calf from May 26, 1928, service

TABLE II—*Data on infected herd, January 1, 1924, to January 1, 1929—Continued*

Cow	Age	Services	Living Calves	Abortions	Dead Calves	Retained Placentas	Present Breeding Condition	Remarks
24	2 yrs.	Bull						Sold for slaughter, July 22, 1924. A positive reactor. Artificially infected by testicular injection, November 19, 1923
52	3 yrs.	Bull						Sold for slaughter, Oct. 14, 1925. A positive reactor. Artificially infected by testicular injection, April 18, 1924
58	2 yrs. 3 mos.	Bull						Negative. Sold for slaughter, Dec. 16, 1926
84	2 yrs.	Bull						Negative. Sold for slaughter, May 1, 1928
93	2 yrs.	Bull						Positive. Now in herd. Becoming negative

were taken by heifer calves raised in the herd. The endeavor has been to keep as young and susceptible a group as possible. The sterile animals and those which could not be demonstrated as spreaders were eliminated from the infected group. New cases of infected animals were added to this herd, as occasion permitted.

DISCUSSION

We stated[1] in 1926: "The data presented seem to indicate that the Bang infection does not spread rapidly among two groups of animals kept entirely separate." The results of work for three years more confirm this view. The results here reported give the basis for a rational and economically sound method for the control of disease due to *Bact. abortus* Bang in cattle. The germ apparently is not long-lived outside the animal body nor is it easily carried from one place to another by attendants. We believe that physical contact, especially licking, is the principal way by which this infection is conveyed from animal to animal in the barn and paddock. In the pasture, the aborted fetus and resultant contaminated food also become important features.

We believe that the pasture and paddock offer more opportunities for the disease to spread than the barn and stanchion. In this connection we desire to condemn heartily the practice of constructing an open feed-manger, such as is being used in some of our modern barns. *No* opportunity should be allowed for attendants to walk in or otherwise contaminate the manger of cattle. The individual manger is most sanitary, but least convenient for the attendant. At least mangers should be especially constructed so as to prevent danger from disease contamination brought in on the feet by careless attendants.

The heifer calves from the infected herd have, with few exceptions, been placed in the clean herd. Over 90 per cent of the calves born from infected dams will become negative to the blood tests at 3 to 6 months of age. This offers an opportunity to build up a clean herd from infected individuals.

TABLE III—*Summary*

CLEAN HERD 33 FEMALES	INFECTED HERD 16 FEMALES
148 services for 87 pregnancies. Ratio 1.7 to 1.	103 services for 43 pregnancies. Ratio 2.4 to 1.
56 living calves for 64 terminated pregnancies (87.8%)	20 living calves for 38 terminated pregnancies (52.6%)
3 abortions for 64 terminated pregnancies (4%)	17 abortions for 38 terminated pregnancies (44%)
4 retained placentas for 64 terminated pregnancies (6%)	18 retained placentas for 38 terminated pregnancies (47%)

The data presented in table III leave no room for doubt as to the economic value of the two herds. Thirty-three per cent more living calves were obtained in the clean herd in the terminated pregnancies. Other comparative data are shown in the table. There seemed to be a very distinct connection between the Bang infection and retained afterbirth. Certain other investigators have failed to find this correlation.

This plan of segregation, based on *no physical contact*, has been tried in several herds in different parts of Minnesota and has been found to give excellent results. It is surprising how quickly one can clean up a badly infected herd by means of the blood tests and segregation, providing physical contact is avoided, and reasonable care is exercised to prevent the spread of the germs. Nearly every cattle-owner can do something to allay the spread of the disease in his herd. This will vary to a high degree. On

the one hand is an owner with two farms a mile apart, on which the two groups of cattle can be kept. On the other hand is the small farmer, with only one barn and one pasture. This latter person can have his animals tested and learn which are the dangerous individuals. Some of these are unprofitable from the start and should be disposed of. Again, this individual can keep his young stock separate from the older animals, even if this has to be done in the same barn or in the horse-barn and by separate pastures. If the cattle-owner says there is nothing he can do to prevent the spread of the disease, veterinarians should point out the specific things he can do and help him in carrying them out. That this is not always easy is very true. It is as true today as yesterday, "There is no royal road to success." The knowledge is now available which enables us to adopt a rational and economically sound policy for the control of infection due to *Bact. abortus*.

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Permanent headquarters of A. V. M. A., Book Building, Detroit; temporary headquarters of all live-wire veterinarians, Detroit, August 13-16.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

SALMONELLA AERTRYCKE INFECTION IN THE CANARY BIRD

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During the last year, five outbreaks of an epizootic occurring in canary bird stores throughout the state of Michigan have been called to our attention. The disease was of a highly contagious character and a large number of birds had died in each case before dead birds were sent to this laboratory for diagnosis.

Cultures made from the heart and liver of some of the first birds sent to us yielded a pure culture of an organism which was later identified as *Salmonella aertrycke*. This organism was differentiated from *Salmonella schotmulleri* on the medium perfected by Jordan and Harmon.[1]

Six birds were purchased and exposed to the infection by contaminating the drinking water with one-half cubic centimeter of a broth culture of the organism.

The incubation period of the disease produced ranged from four to five days. The symptoms produced in the birds were practically identical with those described by Beaudette and Edwards[2] in their work with canary birds when dealing with the same infection: "The first symptom of the disease was a 'puffing up' of the birds. There were some indications of constipation and later diarrhea developed, the droppings being greenish in color. The droppings in some instances were decidedly bloody. During the last stages large amounts of urates usually appeared in the excreta." The course of the disease proved to be very rapid as all of the birds died in from two to four days after the first symptoms of "puffing up" appeared.

The macroscopic lesions were very similar to these described by the above workers: "Catarrhal enteritis with reddening of the mucosa was present. The intestinal contents were bloody in

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some instances. Congestion appeared in the liver, lungs and kidneys. The spleen was not visibly affected."

A microscopic examination of the tissues of the liver, lungs, kidneys, spleen and intestines was made in all cases. All of the birds showed necrotic enteritis, marked congestion of the liver, and congestion of the kidneys with extensive necrosis of the tubular epithelium. The lungs were in a general state of congestion and teeming with organisms. The organisms in these cases measured 0.8 to 1.0 by 1.5 to 3.5 microns, many of them having extremely rounded ends and bulging in the center. The same organism cultured on standard laboratory media measured 0.4 to 0.5 by 1.0 to 2.5 microns. In four of the birds there were extensive foci of necrosis in the lungs, surrounded by extreme hyperemia and cellular infiltration.

One very interesting feature was the ease with which the organism could be isolated in pure culture from the infected birds. Brilliant-green liver-agar medium, as perfected by Mallman, Thorp and Semmes[3] and which is used in this laboratory in routine isolations of *Salmonella pullorum*, was used. The heart, liver, kidneys, lungs, spleen and feces yielded pure cultures in all of the test birds, with the exception of one, in which we were unable to isolate the organism from the kidneys. Pure cultures also were obtained from the above organs of dead birds sent to the laboratory for diagnosis.

One group of three dead birds received for diagnosis consisted of females, each of which contained two ova about the size of a large pea. The organism also was isolated in pure culture from all of these ova.

Conclusions

1. The incubation period of *S. aertrycke* infection in the canary bird is four to five days.

2. The course of the disease extends over a period of two to four days.

3. The organism can be isolated in pure culture from the liver, spleen, heart, lungs, kidneys and ova, and from the feces when brilliant-green medium is used.

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For the period up to and including 1917 we listed 154 references on page 20 of the 21st Annual Report of the United States Live Stock Sanitary Association: for 1918, 21 references on p. 150 of the 22nd Annual Report; for 1919, 36 references on p. 123 of the 23rd Annual Report, for 1920, 52 references on p. 96 of the 24th Annual Report; for 1921, 44 references on p. 48 of the 25th Annual Report. Since 1921 the Journal of the A. V. M. A. has printed the bibliographies as follows: For 1922, 54 references, p. 398, lxiv, December, 1923; for 1923, 76 references, p. 532, lxvi, January, 1925; for 1924, 57 references, p. 238, lxviii, November, 1925; for 1925, 96 references, p. 393, lxx, December, 1926; for 1926, 96 references, p. 114, lxxii, October, 1927. There has been evidence of a growing interest in the subject. This year the number of references is slightly below the number for the past two years. Diagnosis, immunization and the relation of Bang's disease to Malta fever are the chief topics.

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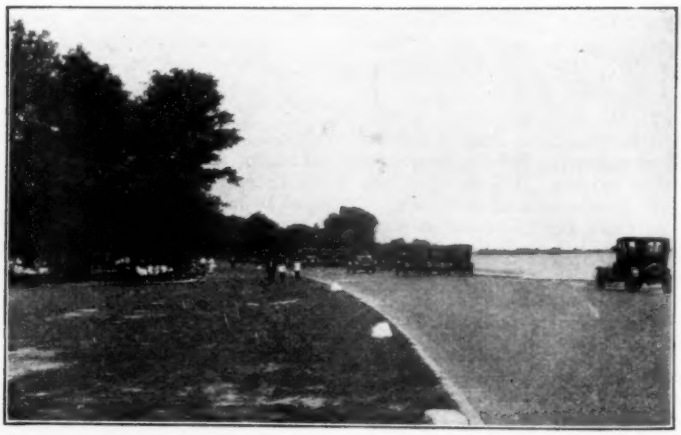
A steamship passes Detroit every three minutes during the season.



Scene on Belle Isle, Detroit

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Colonel Ray J. Stanclift is relieved from duty at Fort Sam Houston, Tex., effective on or about August 10, 1929, and assigned to Fort Riley, Kans., for duty.

Lt. Colonel Robert C. Musser is relieved from duty at the Army Veterinary School, effective on or about August 1, 1929, and assigned to duty at the Chicago general depot, Chicago, Ill.

Lt. Colonel A. L. Mason is relieved from duty at Fort Bliss, Texas, effective on or about August 10, 1929, and directed to report for duty at Fort Sam Houston, Texas.

Orders directing Captain John R. Ludwigs to report to the Army Veterinary School for duty as student have been revoked.

Captain Mott Ramsey is relieved from duty at Fort Ringgold, Texas., and assigned to Fort Sam Houston, Tex., for duty.

Captain Josiah W. Worthington is relieved from further duty at Fort Sam Houston, Tex., effective on or about August 15, 1929, and assigned to Madison Barracks, New York, for duty.

Captain Ralph B. Stewart is relieved from further duty at Fort Riley, Kans., effective on or about July 15, 1929, and assigned to duty at Carlisle Barracks, Pa.

Captain Irby R. Pollard is relieved from further duty at Carlisle Barracks, Pa., effective on or about July 15, 1929, and directed to report to the commanding officer, Quartermaster Depot, Fort Robinson, Nebr., for duty.

Orders relieving Captain Frank C. Hershberger from duty as transport veterinarian on the U. S. transport "Meigs," and from further duty at Fort Mason, Calif., effective on or about July 5, 1929, are amended so as to relieve him on or about September 6, 1929.

Captain Claude F. Cox is relieved from duty at Fort Sam Houston, Tex., effective on or about July 1, 1929, and assigned to duty at Fort Hoyle, Md., for duty.

Major George H. Koon is relieved from duty at Fort Leavenworth, Kans., and assigned to duty as commandant of the Army Veterinary School, Army Medical Center, effective on or about August 10, 1929.

The retirement of Colonel Robert Vans Agnew from active service on May 28, 1929, for age, is announced.

Reserve Corps

By direction of the President, Lt. Colonel Nelson Slater Mayo, Veterinary Corps Reserve is, with his consent, ordered to active duty, effective July 1, 1929. On that date he will proceed from Highland Park, Ill., to Washington, D. C., reporting in person upon arrival to the Surgeon General for training. He will be relieved from duty in time to enable him to arrive at his home on July 14, 1929, on which date he will revert to inactive status.

Promotions

Conroy, James Gibbons, Walkersville, Md.—to Major.
Denman, Clarence B., 273 Hudson Ave., Newark, O.—to Captain.
Goodman, Albert A., Norwood, Colo.—to Captain.
Harbaugh, Fred G., Lubbock, Texas—to 1st Lieutenant.
Rundle, Thomas T., Camarillo, Calif.—to Major.

New Acceptances

Adamson, Cecil Temple	2nd Lieut.	Centerville, Iowa.
Bailey, William W.	2nd Lieut.	Red Lodge, Mont.
Barry, James R.	2nd Lieut.	Norwalk, Wisc.
Bark, Hurdman W.	2nd Lieut.	Sandwich, Ill.

Beretta, Edward H.	2nd Lieut.	Red Lodge, Mont.
Bolin, F. M.	2nd Lieut.	Route No. 4, Carthage, Mo.
Carter, George S.	2nd Lieut.	3008 West St., Ames, Ia.
Lewis, Henry Vance	2nd Lieut.	Edgewood, Iowa.
Johnson, Percy Carl	2nd Lieut.	Cannon Falls, Minn.
McCauley, Earl Dale	2nd Lieut.	Lake Park, Iowa
Mericle, Robert Bruce	2nd Lieut.	Blue Earth, Minn.
McClung, Hugh Edward	2nd Lieut.	R. 3, Hayward, Calif.
De Camp, Daniel	2nd Lieut.	Woodbine, Kansas.
Mohri, Ralph Wm.	2nd Lieut.	Manhattan, Kans.
Moore, Needham Branch	2nd Lieut.	Kinston, N. C.
Omer, Charles Robert	2nd Lieut.	Mankato, Kansas.
Thom, Edwin G.	Captain	Kenosha, Wisc.

Transferred to Auxiliary Reserve, June 20, 1929—Lt. Colonel Levi Enos Day.

The people of Detroit own their own street-car system.

AT FORT SNELLING

The 25th Veterinary Evacuation Hospital was ordered to report for training at Camp Snelling, Minnesota, July 7 to 20, 1929. According to Dr. L. R. Smith, a member of the Unit, this is the first time that an independent veterinary unit of the Reserve Corps has been ordered out for training.

Thirty-five veterinarians comprised the organization, which was highly commended, by the commanding officer of the camp, for the efficiency shown by the unit. It is quite likely that other such units will be ordered out for future camps.

In the photograph on the opposite page are shown thirty of the veterinarians at Fort Snelling.

Front row (left to right): Lt. I. C. Munger, Jr.; Lt. C. A. Ashby; Lt. L. O. Fish (Ind. '16); Lt. R. S. Walden (K. C. V. C. '17), Lennox, S. Dak.; Lt. R. K. Hawley; Lt. D. W. Wright; Lt. D. H. Bibens (K. C. V. C. '11), Chicago, Ill.; Lt. E. M. Brady (Iowa '12), Spencer, Iowa; Capt. C. H. Hart (Chi. '11), South St. Paul, Minn.

Middle row: Lt. Col. L. R. Smith (K. C. V. C. '10), Sioux City, Iowa, Commanding Officer; Capt. H. W. Tornow (K. C. V. C. '09), Wichita, Kans.; Lt. M. L. Dietrich (St. Jos. '22), Newton, Kans.; Capt. W. L. Williamson (O. S. U. '15), Austin, Minn.; Lt. W. G. Lashbrook; Maj. O. C. Selby (Chi. '08), Mankato, Minn., Executive Officer; Lt. F. A. Bonnstetter (Iowa '24), South St. Paul, Minn.; Maj. W. E. Muldoon (Corn. '13), Flora, Ind., Plans and Training Officer; Lt. A. L. Born (Iowa '11), Story City, Iowa.

Rear row: Capt. E. F. Kallenberg (McK. '17), Cedar Rapids, Iowa; Capt. R. A. Moye (Iowa '17), Manchester, Iowa; Lt. R. S. Norton (Chi. '10), Velva, N. Dak.; Lt. R. A. Telford (St. Jos. '17), Waterloo, Iowa; Lt. J. A. Teie (Chi. '12), Hatton, N. Dak.; Lt. C. W. Picht (Colo. '20), St. Paul, Minn.; Lt. L. E. Dietrich, (K. C. V. C. '17), Wichita, Kans.; Capt. F. Low (O. S. U. '17), Oakes, N. Dak.; Capt. J. O. Schlegel (K. C. V. C. '11), Sioux City, Iowa; Maj. J. G. Noble; Maj. F. B. Croll (K. C. V. C. '12), Capt. R. D. Wall (Chi. '09), Des Moines, Iowa.

Absent when picture was taken: Capt. A. C. Barr (K. C. V. C. '06), Kansas City, Mo.; Maj. Geo. E. Bartholomees (K. C. V. C. '11), Sheldon, Mo.; Capt. Julius Stotchik (Corn. '16), Galesburg, Ill.; Capt. L. E. Willey (Iowa '11), Sioux City, Iowa; Maj. F. J. Younglove (McK. '16), Bemidji, Minn.

25TH VETERINARY EVACUATION HOSPITAL AT FORT SNELLING

AT CARLISLE BARRACKS

Fourteen veterinarians, all members of the Reserve Corps, U. S. Army, were on active duty at Carlisle Barracks, Carlisle, Pa., for a training period of two weeks, from July 7 to 20, 1929. The group is shown in the photograph on the opposite page.

Front row (left to right): Capt. L. A. Paquin (McGill '98), Webster, Mass.; Capt. Elmer Lash (K. C. V. C. '07), Washington, D. C.; Maj. James G. Conroy, Walkersville, Md.; Capt. Robert W. McKibbin (Ont. '95), Waynesboro, Pa.; Maj. R. S. Sugg (A. P. I. '15), Auburn, Ala.; Maj. H. W. Schoening (U. P. '07), Washington, D. C.; Capt. N. J. Pyle (U. P. '18), Amherst, Mass.; Capt. E. L. Hannon (Chi,. '07), Pittsfield, Mass.

Rear row: Lt. C. C. Rife (Corn. '25), Altanta, Ga.; Capt. C. P. Marcus (K. C. V. C. '17), Nutley, N. J.; Capt. Paul C. Kucher (K. C. V. C. '16), Fort Wayne, Ind.; Maj. Ralph B. McCord (Corn. '13), Northeast, Pa.; Capt. Charles V. Morris (Corn. '17), Cambridge, Mass.; Lt. John W. Burke (O. S. U. '23), Dayton, Ohio.

The theatres and motion-picture houses in Detroit have a seating capacity of almost 200,000.

Horses in London

According to a clipping from a London newspaper, the equine population of London has been increased by some thousands during the last few years. The reason is said to be a purely economic one, due to the fact that people are beginning to realize that the use of horses under modern conditions is more of a paying proposition than it used to be.

The introduction of the eight-hour working day is given as one reason. It is claimed that previously horses were usually worked beyond the limit of maximum efficiency. Now the horses share the shorter hours with the men. Cases of colic have been reduced to one-fifth the former number; lameness is less common and the average period of usefulness of the horse has increased 50 per cent.

Three automobiles out of every four in the United States are manufactured in Detroit.

The Art Center, Detroit

VETERINARIANS AT CARLISLE BARRACKS

MISCELLANEOUS

Abortion-Free Herds in Oregon

On May 22, 1929, the Oregon State Live Stock Sanitary Board issued its first state certificates for herds of cattle free of infectious abortion. These certificates were handed out by Governor I. L. Patterson to the owners of the herds, at Albany, Oregon, in conjunction with the annual spring show of the Linn-Benton Jersey Cattle Club. This show was one of a series of six which are held each spring. These shows are one-day affairs and include cattle from one or two counties. In this particular instance the counties of Benton and Linn were included. There were 110 cattle on exhibition from twenty-three herds. All of these animals were not only abortion-free themselves, but came from herds which have been tested for abortion and found free of the disease. As far as available information goes, this was the first abortion-free show to be held in the Northwest. The requirement that the animals exhibited be from abortion-free herds was entirely voluntary on the part of the members of the Club holding the show.

Benton County is the home of the Oregon State Agricultural College. Linn County is just across the river from the College, being only a mile from the campus. The experimental work with the field control of infectious abortion, conducted by the College, has naturally been more or less concentrated in these two counties because of their proximity to the laboratories at Corvallis. The first work was started in Linn County, more than ten years ago. As the number of clean herds gradually increased, the members of this cattle club found an increasing number of herds were refusing to show because of the possible danger of infection. When the directors decided to require that all animals exhibited come from abortion-free herds, they felt that they would not have so many animals on exhibition as they have had in the past. As a matter of fact, the show held this year was the largest one ever held. Considering that all of the animals exhibited were of the Jersey breed, the show was rather remarkable in this respect. It is quite likely that only the largest of the state fairs held throughout the country this year will have as many Jerseys on exhibit.

Fish and Bait

At the request of Dr. John H. Winstanley, of Philadelphia, we are reproducing a paragraph from the Philadelphia *Inquirer*, of July 13, 1929. and a photograph (fig. 1). The latter is submitted as documentary evidence of the statement taken from the Philadelphia paper.

FIG. 1. Fish.

"Dr. Roger S. Amadon has every right to be satisfied with his Sunday's outing on Delaware Bay. He fished from a small rowboat and caught three black drum fish weighing in the aggregate 167 lbs."

Dr. Amadon is professor of physiology in the Veterinary School, University of Pennsylvania. When it is too hot for studying the antics of the ruminant digestive apparatus, he very frequently deserts the manometer and kymograph for the rod and reel.

FIG. 2. Bait.

In commenting on the fish which were shown on page 85 of the July issue of the JOURNAL (fig. 2), Dr. Winstanley, who makes some claims of being a follower of Izaak Walton, went on to say that he uses fish of that size for bait. Well, Dr. Palmer, it's your turn now.

DR. VERANUS A. MOORE

Dean Moore Retires

Dr. Veranus A. Moore retired as Dean of the New York State Veterinary College at Cornell University, July 1, 1929, and has been succeeded by Dr. Pierre A. Fish, who has been a member of the Cornell faculty since 1890.

Dr. Moore served as dean for the past twenty-one years and was a member of the original faculty when the Veterinary College was founded in 1896. Prior to going to Cornell, Dr. Moore was in the service of the U. S. Bureau of Animal Industry, at Washington.

The annual meeting of the New York State Veterinary Medical Society was held at Ithaca, June 19-20, and the program was concluded with a dinner in honor of Dean Moore. An impressive part of the program was the presentation, by Professor Simon H. Gage, to Dean Moore, of an illuminated scroll bearing the signatures of a large number of his associates and friends.

At a meeting of the Veterinary College faculty, the following resolution was adopted:

Resolution

WHEREAS, The age of retirement from the active duties of administration and instruction has been reached by Veranus A. Moore and

WHEREAS, His faithful and exemplary service as head of the Department of Pathology and Bacteriology for twelve years, and his conscientious and effective service in the higher office of Dean of the New York State Veterinary College for twenty-one years has resulted in the development of an institution second to none in research and instruction in veterinary subjects and has assisted materially in placing the veterinary profession on a plane with other learned professions and

WHEREAS, his devotion to the insistence on high ideals has been a potent factor in strengthening the College and uplifting the profession and

WHEREAS, his modest and unselfish personal attributes have endeared him to his colleagues, to the alumni and students, and to the profession at large, be it

Resolved, That we, his colleagues in the faculty, assure him of our appreciation of the invaluable service he has rendered in the fields of veterinary education, animal industry, and public health, and that his indefatigable energy and sound judgment has brought honor and prestige to the College, thereby reflecting credit upon his associates and be it further

Resolved, That while we regret his retirement from the official duties of the College, we trust his wise counsel and advice may still be available in the years to come. We further express the hope that his future may be peaceful and serene and he may feel that the burdens he has borne are compensated for, in part, by the respect and admiration of his friends. We are grateful for the example of his life; his career of usefulness and unselfishness, and assure him that, in our opinion, the world is better for his having lived in it.

The portrait of Dr. Moore, shown on the opposite page, was presented by the alumni of the Veterinary College to Cornell University.

Pamphlet and Poster to Protect Porkers

"Are You Paid or Do You Pay?" is the title of an interesting pamphlet published by the Corn States Serum Company, of Omaha, Nebr., in the interests of the swine industry. The contents of the pamphlet consist largely of practical pointers picked from quite a wide variety of publications and reports, papers, radio talks, etc., all very much to the point and planned to promote prompt, periodical and proper protection for porkers. Prompt protection means early vaccination—as soon after weaning as possible. Periodical means year in and year out—every year. Proper protection is given only when the immunizing process is performed by a graduate veterinarian with products that have been painstakingly prepared.

Through the medium of a poster, also prepared by the Corn States Serum Company, some of the same pointers are emphasized. "Have Your Pigs Vaccinated Now" and "Delay is Both Dangerous and Expensive" are two thoughts that are driven home with a punch. If you have not seen copies of either the pamphlet or poster, ask the Corn States Serum Company to send you copies. In addition to promoting prophylaxis for porkers, both pamphlet and poster will be productive of some powerful publicity for the profession.

One Detroit firm uses 600 horses in delivering its products throughout the city and environs.

ALL SET FOR DETROIT
According to Dr. K. J. Moye, Keytesville, Mo.

Itinerary of Official Tour

The following itinerary has been announced for the official tour of Europe in connection with the Tenth International Veterinary Congress, to be held in London, England, August 4-9, 1930:

June 14	New York	Sail on one of the palatial cabin steamers.
June 21-27	Paris	Visit to the famous Veterinary School at Alfort. Visit of the Pasteur Institute and laboratories. Abattoirs and stockyards of La Villete. Sightseeing in Paris and environment, including motor trips to Versailles, Fontainebleau.
June 28	Marseilles	via Avignon.
June 29-30	Nice, Monaco and Monte Carlo	Wonderful "Corniche Drive". Famous casino.
July 1-2	Milan	Veterinary College and city abattoirs, including visit to the beautiful Cathedral of Milan.
July 3-4	Venice	St. Mark's Cathedral and sight-seeing through the canals in gondolas.
July 5	Bern	Serum Institute and Veterinary College. Sight seeing.
July 6-7	Interlaken	Two days in the beautiful valley of the Jungfrau, Scheidegg and Grindelwald.
July 8-9	Lucerne, Rigi	The finest Swiss scenery.
July 10	Zurich	Veterinary College, Etc.
July 11-12	Munich	Veterinary College. Sight-seeing. Center of Art and Music.
July 13-15	Vienna	Veterinary College, Hygienic Institute. Sight-seeing. (A second Paris).
July 16-17	Budapest	Veterinary College, city market. Sight-seeing.
July 18	To Dresden	A day crossing Austria and Czechoslovakia.
July 19-20	Dresden	Visit of the art galleries and city sights.
July 21	Leipsic	Most modern Veterinary School, abattoirs and stockyards in the world.
July 22-24	Berlin	Veterinary College, city sights, abattoirs and stockyards. Imperial Board of Health Laboratories. Visit to Potsdam, via Havel Lakes.
July 25	Hannover	Veterinary College.
July 26	Wiesbaden and Frankfort	A beautiful resort and another interesting city.
July 27	Mainz	Rhine steamer trip. A delightful day.
July 28	Cologne	Castles, Legends, Romances.
July 29-31	Utrecht, The Hague, Amsterdam	Where there will be an opportunity to see how the Dutch keep their land from being overflowed with water, visiting the dikes and locks, and the famous art galleries. Visit of the Island of Marken and Volendam.
Aug. 1-2	Brussels	Veterinary College. Sight-seeing.
Aug. 3	Ostende	Europe's greatest seaside resort.
Aug. 4-9	London	Attending the International Veterinary Congress
Aug. 10-17	England and Scotland	Delightful trips are being planned for this time in England and Scotland.

Inquiries in connection with business arrangements should be addressed to The Beaux-Arts Tours, 2030 15th St., N. W., Washington, D. C.

ASSOCIATION MEETINGS

CALIFORNIA STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the California State Veterinary Medical Association was held at Sequoia National Park, June 10-11-12, 1929. The meeting was held in Giant Forest Lodge, at an elevation of 6400 feet. Those in attendance soon found that the climatic conditions in the mountains were slightly different from those in the valley below.

Dr. E. H. Barger, of Berkeley, presided and, after calling the meeting to order, introduced Col. John R. White, superintendent of Sequoia National Park, who gave the address of welcome. Col. White stated that he thought that the weather was ideal for a veterinary meeting. This caused considerable laughter, as the night previous a heavy storm occurred in the mountains accompanied by thunder and lightning. The rain later turned to hail and snow and when we arose in the morning the ground was covered with nearly an inch of snow and hail. Mr. H. E. Williams, field representative of the Secretary of the Interior and formerly commissioner of agriculture of West Virginia, also spoke briefly. Mr. Williams stated that he had had considerable experience with veterinarians in the East during an outbreak of foot-and-mouth disease and that this former contact with veterinarians had been a very agreeable one. Dr. O. A. Longley, of San Francisco, responded to the address of welcome. Dr. Longley remarked that Col. White was a very good story teller, after he (Col. White) had just described the perfect weather for a veterinary meeting.

In his presidential address Dr. Barger mentioned the work of the Membership Committee during the past year in securing so many new members for the A. V. M. A. He also commented on the record made in disposing of foot-and-mouth disease in Los Angeles County during the early part of the year.

The literary program was opened with a paper by Dr. Jacob Traum, of the University of California, entitled, "Results of Recent Research in Foot-and-Mouth Disease." Dr. Traum gave a history of the work that has been done by laboratory workers on foot-and-mouth disease. He stated that no sure method of immunization has been discovered to date and re-

ported that vaccine will protect guinea pigs in some instances. He stated also that serum has been found to be quite effective in the treatment of foot-and-mouth disease in Europe.

Dr. H. P. Bonnikson, of the California State Department of Agriculture, presented a paper, entitled, "Ship Inspection a Factor in Animal Disease Control." (This paper will be published in the JOURNAL.) Dr. Bonnikson brought out the fact that it is quite certain that the outbreak of foot-and-mouth disease that paid California a rather lengthy visit in 1924, as well as the much shorter visit this year, had been brought to California by garbage that had been taken from ships that had come from or touched, during their voyages, a country that was infected with foot-and-mouth disease.

The program Monday afternoon was opened by Dr. C. H. Reid, of Hollywood, who read a paper, entitled, "Apotheosis of Veterinary Medicine." (This paper will be published in the JOURNAL.) Dr. Reid pointed out that times are rapidly changing in the practice of veterinary medicine and that great progress has been made in our profession during the last few decades.

Dr. J. P. Iverson, Chief, Division of Animal Industry, State Department of Agriculture, was the next speaker. His subject was "A Brief Review of New Legislation of Interest to Veterinarians." Dr. Iverson opened his talk by stating that Assembly Bill 739, creating a Department of Professional and Vocational Standards, had been signed by the Governor. While there is some dissatisfaction over the law, due to the classification received by veterinarians, it appears that investigators employed by the new department are supposed to prosecute all violators in the class where veterinarians are placed. The new Bovine Tuberculosis Bill was then discussed by Dr. Iverson. The discussion disclosed the fact that while $100,000.00 was appropriated to indemnify owners of the slaughtered animals, Dr. Iverson also informed us that this new law may be unconstitutional. If this should prove to be the case, the Constitution of the State will have to be amended before owners of tuberculous animals may receive compensation.

Dr. Iverson next touched on a proposed law that would make the cutting of ears and tails of dogs unlawful. This bill was introduced in the Senate, passed the House, but was defeated in the Assembly. Dr. Iverson next mentioned the two new state meat inspection laws. One of these proposed laws failed to pass, while the Crowley Bill passed, but up to the present time has not

been signed by the Governor. The so-called Veronal Law, "An Act to Add a New Section 347½ to the Penal Code Relating to the Sale and Use of Poisons," was next read by Dr. Iverson. From the wording of this law, it appears that California veterinarians will be deprived of the privilege of dispensing veronal, barbitol, or any of the derivatives of diethylbarbituric acid, although veterinarians may purchase these drugs either at retail or wholesale without prescriptions and apparently may also administer these drugs in their practices. It appears that veterinarians have been discriminated against in the passage of this law, probably unintentionally.

Other laws mentioned by Dr. Iverson as having been passed at the recent session of the Legislature and signed by the Governor include laws regulating the improper labeling of drugs and one barring refuse and garbage from vessels that have touched foreign countries that are infected with foot-and-mouth disease and later enter California ports. The Legislature also provided for the indemnification of owners of animals slaughtered in the recent outbreak of foot-and-mouth disease.

It was moved by Dr. J. P. Bushong that a resolution be drafted by the Resolution Committee requesting the Governor to sign two bills and veto one bill before him. This motion was seconded and carried.

The next speaker was Dr. L. O. Henrich, of Corcoran, who opened a discussion on "Policy." Dr. Henrich mentioned radio broadcasting as a means of advertising the veterinarian and his work. The speaker also stated that in some instances information concerning the care and treatment of live stock is broadcast by men who are not qualified to give such information. Dr. Henrich is of the opinion that radio broadcasting describing the work of the veterinarian is legitimate if properly conducted. A lengthy discussion on policy and advertising followed Dr. Henrich's remarks.

The Monday afternoon program was brought to a close with a discussion of the proposed changes in the By-laws of the Association. Dr. Traum, as chairman of the Special Committee, read the new by-laws as written by the Committee. With the exception of one item, referring to the admission of new members, the Association adopted the proposed by-laws. One provides for the addition of a second and a third vice-president to the list of officers. Under the new by-laws, the first vice-president is ex-officio chairman of the Executive Committee; the second vice-

president, chairman of the Committee on Program; and the third vice-president, chairman of the Committee on Membership and A. V. M. A. Affairs.

Monday evening, two motion pictures were shown through the courtesy of the State Fish and Game Commission. One was entitled "Deer and Their Kin" and the other "Trout Culture." Dr. H. Van Roekel, of the Hooper Foundation for Medical Research, San Francisco, delivered a lecture in connection with the second picture.

Dr. A. G. Boyd, of the State Department of Agriculture, opened the program Tuesday morning with a paper, entitled, "Bovine Bacillary Hemoglobinuria." Dr. Boyd stated that this disease has been found recently in South America and in several of our western states, including Oregon, Washington, Nevada and California. The speaker stated that the disease is found usually in the summer and early fall and that the mortality is often as high as 95 per cent. The Nevada Experiment Station has studied this disease extensively during recent years and has prepared a vaccine for use as a prophylactic and a serum that is 75 per cent effective as a curative agent when treatment is administered early. The discussion which followed centered on the differential diagnosis between hemoglobinuria and hemorrhagic septicemia in adult cattle.

Dr. J. P. Bushong, of the Los Angeles Medical Milk Commission, spoke on his experiences in abortion control work. Dr. Bushong's work is confined to certified dairies and during the past two years he has bled 22,000 dairy animals in connection with locating those infected with abortion. After January 1, 1930, all dairy herds producing certified milk in Los Angeles must be practically free from abortion. Dr. Bushong stated that two reasons are advanced for eradicating abortion from dairy herds. First, the possibility of producing undulant fever in the human and, second, from a purely economic standpoint.

Public health officials state that nearly 800 cases of undulant fever in humans have been found in the United States, believed to have been caused by the consumption of milk infected with the abortion organism. From an economic standpoint, it is pointed out that herds free of abortion will produce more calves and more milk than herds that are infected.

Dr. J. R. Beach, University of California, Berkeley, presented "Present Status of Methods for the Diagnosis of Bacillary White Diarrhea." Dr. Beach described methods of making the pullorin

and agglutination tests and stated that he had recently used three different types of pullorin. By the use of a chart, Dr. Beach showed a summary of the tests in which the different types of pullorin were used, compared with the agglutination test and bacteriological findings. The results of Dr. Beach's experiments apparently indicate that the pullorin test is not so reliable at present as the agglutination test.

The business session, originally scheduled for Monday evening, was held Tuesday afternoon, at which time regular routine business was conducted and the annual election of officers was held. This resulted as follows: President, Dr. W. L. Edwards, Visalia; first vice-president, Dr. P. C. Guyselman, Fontana; second vice-president, Dr. Rudolph Snyder, Sacramento; third vice-president, Dr. L. E. Pike, Long Beach; treasurer, Dr. D. F. Fox, Sacramento; and secretary, Dr. W. L. Curtis, Los Angeles.

The annual report of the Secretary showed that there were 306 members on the roll, three of whom were dropped during the meeting and eight new members were admitted, bringing the total membership to 311 at the time of adjournment. The annual report of the Treasurer showed a balance of $1,709.42. Dr. Frank Griffith, of Hanford was made a life member.

Following the business meeting, the balance of the afternoon was spent in visiting nearby places of interest in Sequoia National Park. The bear-pits first were visited, later Moro Rock and our last stop was made at the General Sherman Tree. This tree is said to be the largest and oldest of living things. It is 37 feet in diameter at its base and its weight is estimated to be over 6,000 tons. Its age is thought to be between 4,000 and 6,000 years.

Tuesday evening an entertainment was given in the dining-room, followed by a dance. The entertainment was unusually good and, judging from the crowd in attendance, it appeared as though all the people in the park must have been present.

Wednesday morning, the program was opened with a paper by Dr. R. B. Griffenhagen, of Fresno, entitled, "The Veterinarian." The author stated that the number of veterinary students in North America today is small as compared with the number twenty or twenty-five years ago. Dr. Griffenhagen also stated that the general public does not know just what the veterinarian is doing and he also pointed out that formerly nearly all veterinarians confined their work principally to the treatment of horses, while today many veterinarians are employed in public health

service and many private concerns also employ their own veterinarians.

Dr. F. W. Wood, of Berkeley, presented a paper, entitled, "The English Method of Preparing Canine Distemper Vaccine and Virus." He described in detail the method used by Dr. Laidlaw and Major Dunkin in the preparation and use of their biological products for immunizing dogs against distemper. A general discussion followed, showing the deep interest that practicing veterinarians have for information concerning this subject.

Dr. L. E. Pike, of the Los Angeles County Livestock Inspector's office, was the next speaker. His subject was "Diseases of Domestic Rabbits." Dr. Pike stated that the rabbit industry in Southern California is fast becoming an industry of considerable importance. The speaker stated that so-called "snuffles" and coccidiosis are the most common and serious diseases of rabbits. Dr. Pike mentioned that several organisms are credited with being the causative factor in so-called "snuffles." The speaker also mentioned that external parasites are commonly found on rabbits in California, but that internal parasites in rabbits are rather rare.

Dr. J. A. Howarth, of the University of California, Davis, presented a paper, entitled, "Spirochetes as the Exciting Agent in Specific Necrosis and Tumor Formation in Swine." The essayist stated that spirochetes are found frequently in tumors that develop following the castration of swine. He also stated that these organisms are commonly found in many cases of so-called "bull nose" in swine and that possibly they may be the causative factor in this condition.

Dr. J. B. Jones, of Los Angeles, closed the literary program with a paper, entitled, "Canine Rabies." His paper brought out the fact that rabies first appeared in California in 1909, when a dog suffering from the disease was found in Los Angeles, thought to have escaped from an express company. Shortly after, in Stockton, a dog that had escaped from a circus was found suffering from rabies. Since 1909 there have been over 15,000 cases of canine rabies reported in California, and 69 human deaths have occurred during the last twenty years.

W. L. CURTIS, *Secretary*

Detroit—A. V. M. A.
1900—1916—1929

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The nineteenth annual meeting of the State Veterinary Medical Association of Texas met at A. & M. College, College Station, June 17-20, 1929. Dr. Ross Hodges, of Ranger, was elected president for the ensuing year at the business session held in conjunction with the annual short course for graduate veterinarians. Other officers elected were: Dr. T. O. Booth, Temple, first vice-president; Dr. Frank Hecker, Houston, second vice-president; Dr. D. Pearce, Leonard, secretary-treasurer.

Dean E. J. Kyle formally welcomed the veterinarians to A. & M. at the business session, Wednesday evening. He spoke at length on a most vital subject, mainly agriculture and veterinary science, working together today in trying to stabilize and bring about relief to the farmer. Dean Kyle deplored the retrograde of the land and the hopelessness of the problem being solved without the cooperation of veterinary science and agriculture. Dr. N. F. Williams, State Veterinarian, gave the response. Other speakers included Dr. M. E. Gleason, of San Antonio, the retiring president, who delivered the presidential address.

Col. R. J. Stanclift, V. C., U. S. Army, Fort Sam Houston, lectured at length on "The Army Veterinarian and the Practitioner." He spoke of the great need of a basic education in order to be able to succeed and he stated that the A. & M. College was well able to give this. He also gave a demonstration of the steps by which it is possible to become a good judge of a sound horse. Dr. R. A. Self, of Dallas, gave an illustrated lecture on the ten breeds and types of dogs most commonly treated in small-animal hospitals in Texas. Dr. J. H. Wiener, of Kansas City, Mo., gave his familiar topic, "Veterinary Nostrums."

Dr. C. E. Salsbery, of Kansas City, Mo., gave the Association one of the most enlightening and interesting lectures ever prepresented to this body of men on "*Brucella Abortus* and *Brucella Melitensis* Infections and Public Health Problems." He cited ten cases of undulant fever, giving statistics and histories.

The veterinarians appreciated and derived unusual benefit from the series of lectures by Dr. T. H. Ferguson, of Lake Geneva, Wis. Some of the subjects discussed by him were: "Mastitis," "Diseases of the Digestive Tract," "Feeding and Care of Calves" and "Sterility." Dr. Ferguson proved to be a veritable mine of information for the veterinarians of Texas.

Veterinarians in attendance at meeting of State Veterinary Medical Association of Texas, College Station, June 17-20, 1929

Prof. Charles N. Shepardson, of the Dairy Department, gave an interesting talk on the dairy cow and her efficiency as indicated by types and breeds. Other outstanding men who lectured were Dr. Gerard Dikmans, U. S. B. A. I., Jeanerette, La., on "Anaplasmosis of Cattle," and Dr. Hubert Schmidt, of A. & M. College, on "The Newer Developments in the Pathology of Sheep Diseases." Dr. B. W. Boyd, U. S. B. A. I., San Antonio, demonstrated the application of the intradermal and ophthalmic tuberculin tests in cattle and discussed some of the pitfalls of tuberculin testing.

Case reports were made by Dr. V. A. Scott, Stephenville. Dr. T. O. Booth, of Temple, spoke on some important considerations in connection with successful immunization of swine against cholera. Dr. M. E. Gleason, of San Antonio, discussed the importance of the services of veterinarians in maintaining the standard milk ordinance in connection with dairy inspection. Dr. Fred B. Green, of the State Health Department, Austin, discussed and demonstrated some important laboratory tests used in dairy inspection. He was aided by Dr. A. E. Wharton, of A. & M. College.

Dr. A. A. Lenert, of A. & M. College, presided at the afternoon clinic. Dr. R. C. Dunn gave a lecture on poultry diseases. Dr. D. H. Reid, of the Poultry Department, spoke on some of the accomplishments and objectives of poultry breeding.

The short course was brought to a close Thursday with a program dealing for the most part with small-animal hospital management, including common diseases of small animals, surgical methods and feeding. Among those who took part in the program were Dr. R. L. Ray, San Antonio; Dr. W. G. Brock, Dallas; Dr. H. L. Blackburn, Fort Worth; and Dr. T. O. Scott, Waco.

Eighty-eight men registered for the meeting, many of whom were accompanied by their wives. The Ladies' Auxiliary to the Association met at the home of Dr. and Mrs. Mark Francis, Wednesday afternoon, and inducted the recently elected officers. A round of entertainment and pleasure was provided for the ladies, including a bridge party at the home of Mrs. A. A. Lenert; a game tournament, honoring the ladies of the Auxiliary, at the home of Mrs. R. P. Marsteller; and theatre parties given by Mrs. Hubert Schmidt and Mrs. Francis.

D. PEARCE, *Secretary-Treasurer*

Detroit has eleven miles of river frontage.

KANSAS CITY ASSOCIATION OF VETERINARIANS

The Kansas City Association of Veterinarians held its regular monthly meeting at the Baltimore Hotel, Tuesday evening, May 14, 1929.

The program was devoted to the discussion of undulant fever in man and its relation to live stock. Dr. R. L. Laybourn, of the State Health Department laboratories, Jefferson City, Mo., outlined the conditions existing in Missouri as far as the disease has come under his observations. The State Health Laboratory, under the direction of Dr. Laybourn, is taking an active part in investigating different cases and much good information is being assembled.

Dr. F. E. Angle, of the Bethany Hospital, Kansas City, Kans., discussed clinical cases that had come under his observation and also the results of bacterin treatment of persons showing clinical symptoms. Good results have followed the use of the bacterin treatment in most cases.

Dr. C. H. Kitselman, in charge of abortion disease work at Kansas State Agricultural College, gave a short talk on conditions as he has observed them in Kansas.

This was the best attended meeting ever held by the Association. Seventy-nine members and visitors were present. Several physicians and health officials from the surrounding territory were present. This will set a mark for other local associations to "shoot at," for attendance at a regular meeting and nothing but program for attraction.

The Association meets the second Tuesday night of each month. All graduate veterinarians are welcome.

J. D. Ray, *Secretary*

MASSACHUSETTS VETERINARY ASSOCIATION

The June meeting of the Massachusetts Veterinary Association was held in Springfield, June 20, 1929. This meeting was held as a part of the tenth annual Eastern States Conference on the Eradication of Tuberculosis and the program extended over the full third day. It is doubtful if a more interesting and profitable single-day program has ever been staged. In the morning the following papers were presented:

"Immunization of Dogs Against Distemper by the Laidlaw-Dunkin Method," Dr. A. Eichhorn, Director, Veterinary Department, Lederle Antitoxin Laboratories.

"Common Diseases of Poultry," Dr. N. J. Pyle, Pathologist, Department of Veterinary Science, Massachusetts Agricultural College.

"Vincent's Disease or Trench Mouth in Dogs," Dr. Hugh F. Dailey, Chief Veterinarian, Angell Memorial Hospital, Boston, Mass.
"Some Phases of Small Animal Practice," Dr. Wm. J. Lentz, Director, Small Animal Clinic, School of Veterinary Medicine, University of Pennsylvania.

These papers and addresses were of an exceptionally high order, being full of practical information.

After luncheon the afternoon was given over to a clinic and, judging by the remarks heard on all sides, it was the most interesting and profitable ever attended by the veterinarians present. Many surgical operations of the horse were performed by Dr. Wm. J. Lee, School of Veterinary Medicine, University of Pennsylvania. Dr. Lee not only displayed unusual skill and technic as an operator, but proved himself to be a genial and efficient demonstrator, with the faculty of giving clear, pointed replies to all questions.

The subject of sterility and diagnosis of pregnancy was handled by the internationally known Dr. J. F. DeVine, in his usual interesting and efficient manner. A number of bovine uteri had been obtained for Dr. DeVine to use in demonstrating his address and veterinarians who heard him and saw his demonstration at the A. V. M. A. clinic in Montreal, several years ago, well know his ability to handle this important subject. Several sterile cows were on hand for diagnosis and in addition Dr. DeVine operated on what was thought to be a ridgling, but which proved to be a "high flanker." For this operation Dr. Lee demonstrated his method of administering chloral intravenously by gravity.

Postmortem diagnosis of swine diseases was handled by Dr. I. K. Atherton, Inspector in Charge of Hog Cholera Work, Maryland. To those engaged in the control of swine diseases this proved an interesting part of the program.

Surgical operations on the dog and cat were performed by Dr. William J. Lentz, University of Pennsylvania. Among many other operations Dr. Lentz performed a cesarian on a bitch, demonstrating his method of operation without suturing the uterus. The demonstrations and talks of Dr. Lentz were a real postgraduate course for the small-animal men present.

All of the men taking part are to be congratulated on their high-grade work and much credit is due to the local committee of veterinarians in Springfield for the success of the meeting.

H. W. JAKEMAN, *Secretary.*

Detroit is visited by more than 2,000,000 tourists annually.

NECROLOGY

J. L. BROOKS

Dr. J. L. Brooks, of Clinton, Iowa, died in his car while returning from a call, May 10, 1929. He was 64 years of age. Heart failure was the cause of death.

Dr. Brooks was a graduate of the Ontario Veterinary College, class of 1900, and practiced at Clinton ever since his graduation. He was a member of the Eastern Iowa Veterinary Association. He is survived by his widow and one daughter.

O. R. WIMSETT

Dr. Oral Ray Wimsett, of Shelton, Nebraska, was found dead in his car, on the Lincoln Highway, one mile from Shelton, June 10, 1929. He has been dead several hours when found and death was ascribed to heart failure.

Born at Scotland, Illinois, September 12, 1882, Dr. Wimsett attended local schools and then entered the Indiana Veterinary College. After completing two years at the Indianapolis institution, he transferred to the McKillip Veterinary College and finished there in 1908. He practiced for a short time with his brother, Dr. Ira G. Wimsett (Ind. '02), at Beatrice, Nebr., and then located at Shelton. He took a deep interest in civic affairs and was a member of the City Council at the time of his death.

Dr. Wimsett joined the A. V. M. A. in 1917, and was a member of the Nebraska State Veterinary Medical Association. He held membership in the Masonic and Elks lodges. He is survived by his widow, two brothers and two sisters.

IRVING MOLES

Dr. Irving Moles, of Central City, Iowa, died at a Cedar Rapids Hospital, July 3, 1929, after an illness of several months due to typhoid fever and complications.

Born at Central City, Iowa, June 25, 1889, Dr. Moles attended local schools and then entered the Kansas City Veterinary College. He was graduated from this institution in 1914. The same year he located for general practice at Central City with his brother, Dr. C. L. Moles, now of Dike, Iowa. During the

late war, he held a commission as second lieutenant in the Veterinary Corps and was stationed at Camp Greenleaf, Georgia, and later at Camp Meade, Maryland. He was also commissioned in the Veterinary Reserve Corps.

Dr. Moles joined the A. V. M. A. in 1918. He was a member of both the Eastern Iowa and Linn County veterinary associations, having held the office of vice-president in both organizations. He is survived by his widow and one child, his mother and several sisters and brothers.

WILLIAM A. GIFFEN

Dr. William A. Giffen, of Detroit, Mich., died July 3, 1929, following an illness of two years, caused by a heart affection. He was 63 years of age.

Born at Brandon, Ont., Dr. Giffen studied veterinary medicine at the Ontario Veterinary College and was graduated in 1887. He practiced in Detroit for a number of years and then decided to study dentistry. He entered the Dental Department of the Detroit College of Medicine and was graduated in 1901. He rose rapidly in the dental profession and was honored with the presidency of every society of organized dentistry, beginning with the First District (Detroit) Dental Society, until at Los Angeles, in 1924, he was elected president of the American Dental Association, the highest distinction in his profession. He served as president of the Michigan State Dental Society and as president of the Michigan State Board of Dental Examiners. He was a Mason and held membership in many clubs in and around Detroit.

WARREN T. EDWARDS

Dr. Warren Thomas Edwards, of Philadelphia, Pa., died February 26, 1927. He was a graduate of the University of Pennsylvania, class of 1891. For a number of years Dr. Edwards was in general practice at several locations in Delaware and Chester counties, Pennsylvania, and later was employed by the Pennsylvania State Live Stock Sanitary Board, in glanders control work, in and around Philadelphia. During recent years, he had not been actively identified with veterinary work.

W. J. KUPPER

Dr. W. G. Kupper, of Blue Grass, Iowa, died July 14, 1929. He was a graduate of the Chicago Veterinary College, class of

1907. He was a member of the Iowa Veterinary Medical Association.

EUGENE D. SHEVALIER

Dr. E. D. Shevalier, of Escanaba, Mich., died very suddenly at his home, July 10, 1929, following a heart attack resulting from flu.

Born in Virgil, N. Y., May 4, 1867, he attended the Ontario Veterinary College and was graduated with the class of 1889. He practiced at Escanaba continuously for over forty years.

Dr. Shevalier joined the A. V. M. A. in 1911. He is survived by his widow, one daughter and one grandson.

JOHN W. HARSHBERGER

Professor John W. Harshberger, of Philadelphia, Pa., died April 27, 1929, at the age of 60 years. For many years he was professor of botany at the University of Pennsylvania. As a member of the veterinary faculty, he taught botany and biology to the veterinary students for many years and was admired by every student who sat in his classes. In his make-up was to be found every attribute of the successful teacher.

JOHN ALFRED MANDELL

Professor John Alfred Mandell, of New York City, died May 5, 1929, after an illness of two years. From 1894 to 1897 he was professor of chemistry at the New York College of Veterinary Surgeons. At the time of his death he was professor of chemistry in the University and Bellevue Hospital Medical College of New York University.

Dr. Samuel R. Fulton, of Princeton, Iowa, died March 16, 1929. He was registered as a non-graduate practitioner and had been located at Princeton since 1894. In 1914 he retired from practice and assumed the management of the Princeton telephone system, which he purchased. He was a member of the town council for twelve years and served a number of terms on the school board.

Dr. Jacob A. Gates, of Centerville, Ind., died June 23, 1929, at the age of 90 years. He was actively engaged in general practice until about ten years ago.

Mr. Harry E. Whiteman, of Cedar Rapids, Iowa, head of the Howard-Holt Company of that city, died June 16, 1929, as the result of injuries received three days previously, when he fell twenty feet in an elevator shaft. Mr. Whiteman was well-known to the veterinarians of eastern Iowa.

Our sympathy goes out to Dr. L. H. LaFond, of Detroit, Mich., in the death of his father, Henry LaFond, July 16, 1929, from rabies. Mr. LaFond was bitten by a rabid dog on April 23, took the Pasteur treatment, starting April 29, and developed symptoms of the disease July 14.

Michigan is the home of one of the original four-year veterinary schools in the United States.

PERSONALS

MARRIAGES

Dr. J. R. Robb (O. S. U. '28), of Oak Park, Ill., to Miss Lurretia Holmes, of Hopedale, Ohio, on June 22, 1929.

Dr. Frank D. Landon (U. S. C. V. S. '03), of Great Barrington, Mass., to Miss Alice Munro Davis, of Sheffield, Mass., on June 29, 1929, at Sheffield.

Detroit has thirty-five public parks.

BIRTHS

To Dr. and Mrs. A. H. Francis, of Lincoln, Nebr., a daughter, Betty Jean, June 18, 1929.

Detroit has produced in one year 3,420,500 automobiles at a retail value of $2,632,250,000.

PERSONALS

Dr. Martin Van Der Maaten (K. S. A. C. '29) has located at Le Mars, Iowa.

Dr. E. C. Deubler (U. P. '11) has moved from Philadelphia, Pa., to Newtown, Pa.

Dr. Walter L. Nilson (Iowa '29) is located at 812 20th Ave. S., Minneapolis, Minn.

Dr. S. E. Bitler (Ont. '09) has removed from Turbotville, Pa., to Watsontown, Pa.

Dr. J. B. Paterson (Edinburgh '78), formerly of Regina, Sask., is now at Lumsden, Sask.

Dr. N. J. A. Hederen (Chi. '11) has opened a small animal hospital at 3939 Broadway, Chicago, Ill.

Dr. Daniel W. Gates (U P. '25), formerly located at Bellefonte, Pa., has removed to Millheim, Pa.

Dr. Charles J. Heckard (Iowa '98), of Wheatland, Iowa, left for a tour of the Hawaiian Islands on July 9.

Dr. C. G. Kriedler (Cin. '13), of Maysville, Ky., recently purchased a new home at 1033 E. Second St.

Dr. N. J. Taylor (Ga. '25), formerly of Hazelhurst, Ga., has located at Greenville, Tenn., for general practice.

Dr. F. Gunster (San Fran. '15) has requested a change of address from The Dalles, Ore., to Box 374, Multnomah, Ore.

Dr. C. L. Butler (K. S. A. C. '29) is with the New Jersey Bureau of Animal Industry, State Office Bldg., Trenton, N. J.

Dr. F. H. Brown (Ind. '10) has been re-elected secretary of the Indiana State Live Stock Sanitary Board for another term.

Dr. G. W. Closson (K. C. V. C. '05), of Hollywood, Calif., has purchased the practice of Drs. Tyler and Sparks at Pomona, Calif.

Dr. S. H. Exley (Ga. '24), of the Tennessee Department of Agriculture, has been transferred from Lebanon to Lewisburg, Tenn.

Dr. H. M. Corenzwit (U. P. '27), formerly of Rising Sun, Md., is now located in Philadelphia, Pa., at Frankford Ave. & Somerset St.

Dr. M. M. Davis (McK. '15, Ont. '22), formerly of Bloomington, Wis., has removed to Yankton, S. Dak. Address: 408 Spruce St.

Dr. Edw. L. Wilson (Corn. '12) is General Manager of the Sussex Milk and Cream Company, Inc., 110 Hudson St., New York, N. Y.

Dr. Lloyd Darst (Iowa '28), formerly of Peoria, Ill., is now associated in practice with Dr. Fred Fawcett (Chi. '05), at Princeton, Ill.

Dr. W. Reid Blair (Mont. '02), Director of the New York Zoological Park, has been elected a member of the Authors' Club of New York City.

Dr. C. Canion (Texas '25), formerly of Houston, Texas, is now associated with the Veterinary Service Institute, 924 South Haskell Ave., Dallas, Texas.

Dr. C. H. Patterson (Iowa '26) has resigned his position with the Illinois State Department of Agriculture and has resumed practice at Tiskilwa, Ill.

Dr. J. R. Robb (O. S. U. '28) and Dr. A. W. Anderson (O. S. U. '26) have purchased the Avenue Animal Hospital located at 880 Home Ave., Oak Park, Ill.

Dr. C W. King (O. S. U. '13), of Trenton, Ohio, suffered an attack of illness the latter part of June, which necessitated spending some time in a private hospital.

Dr. C. P. Kime (Chi. '95), of Columbia City, Ind., suffered a broken rib recently, when he stumbled and fell as the result of stepping into a small hole in the ground.

Dr. C. R. Donham (Iowa '21) has resigned his position at the Oregon State Agricultural College to accept one at the University of Minnesota, University Farm, St. Paul.

Dr. Reuben Gordon (Corn. '25), of Patchogue, Long Island, N. Y., was returned a winner in a suit brought by neighbors to have his veterinary hospital declared a nusiance.

Dr. H. J. Buebler (Mich. '29) has been assisting Dr. L. A. Wileden (Mich. '13) at Mason, Mich., while the latter was laid up with a broken leg, as reported in the July issue of the JOURNAL.

Dr. Lawrence Avery (U. S. C. V. S. '17), formerly on tuberculosis eradication work at Frederick, Md., has been transferred to the Zoological Division, Bureau of Animal Industry, at Beltsville, Md.

Dr. R. H. Mohlenhoff (Chi. '91), of Cleveland, Miss., accompanied by Mrs. Mohlenhoff, was a visitor in Arcola, Ill., the early part of June. The Mohlenhoffs were residents of Arcola some years ago.

Dr. W. R. Hinshaw (Mich. '23) has resigned his position at the Massachusetts Agricultural College, Amherst, to accept a somewhat similar position in poultry work at the University of California, Davis.

Dr. T. O. Booth (K. C. V. C. '16) has resigned as Laboratory Director of the Texas Live Stock Sanitary Commission, Fort Worth, and has removed to Temple, Texas, where he is engaged in general practice.

Dr. D. J. Meador (Corn. '11), who has been located at Selma, Ala., for a number of years, has accepted a position at Agricultural Agent for the Louisville and Nashville Railroad, with headquarters at Montgomery, Ala.

Dr. K. F. Meyer (Zurich '08), of the Hooper Foundation for Medical Research, San Francisco, Calif., presided at the meeting of the American Association of Medical Milk Commissions held in Montreal, June 24-25, 1929.

Dr. Russell A. Runnells (Mich. '16), of the Virginia Polytechnic Institute, Blacksburg, was elected to graduate membership in the Michigan State College Chapter of Sigma Xi, at the spring term convocation of this honorary scientific society.

Dr. G. C. Kendall (A. P. I. '28), formerly of Andalusia, Ala., is now located at Lewisburg, Tenn., as a member of the firm of Drs. Jones and Kendall. The senior member of the firm, Dr. J. M. Jones (McK. '12), is State Veterinarian of Tennessee.

Dr. W. H. Hilts (K. S. A. C. '18), who has been a veterinary inspector for the Nevada State Board of Stock Commissioners for the past several years, with headquarters at Elko, has been appointed director of the newly created Division of Animal Industry in the State Board of Stock Commissioners, with headquarters at Reno.

Dr. I. Forest Huddleson (Mich. '25), Research Associate in Bacteriology, Michigan State College, well known for his researches on infectious abortion, is spending the summer as a special agent of the United States Public Health Service in southern Europe and northern Africa. He is studying Mediterranean fever in humans, goats and cattle in localities that are presumed to be the native habitat of this organism.

The University of Michigan, now located at Ann Arbor, was established in Detroit in 1817.